AETIOLOGICAL AND CLINICOPATHOLOGICAL STUDY OF ERYTHRODERMA

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CERTIFICATE

Certified that this dissertation titled **"AETIOLOGICAL AND CLINICOPATHOLOGICAL STUDY OF ERYTHRODERMA"** is a bonafide work done by **Dr.AARTHI M,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2010 – 2013. This work has not previously formed the basis for the award of any degree.

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I, **Dr. AARTHI M** solemnly declare that this dissertation titled **"AETIOLOGICAL AND CLINICOPATHOLOGICAL STUDY OF ERYTHRODERMA"** is a bonafide work done by me at Madras Medical College during 2010-2013 under the guidance and supervision of **Prof. K.MANOHARAN, M.D., D.D.,** Professor and Head, Department of Dermatology, Madras Medical College, Chennai – 600 003.

This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of **M.D Degree in Dermatology, Venereology and Leprology (BRANCH – XII A)**

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CONTENTS

INTRODUCTION

Erythroderma (also known as "Exfoliative dermatitis," "Dermatitis exfoliativa") is a generalized inflammatory disorder of the skin manifesting with erythema and scaling affecting more than 90% of the skin surface.[1] Primary erythroderma arises on normal looking skin due to an underlying systemic disorder or because of a drug reaction, while secondary erythroderma arises from a pre existing dermatoses.

Erythroderma is a morphological reaction pattern of skin having innumerable underlying causes which include preexisting skin conditions like psoriasis, atopic dermatitis, contact dermatitis, systemic skin conditions including malignancy and drug reaction. Even a thorough clinical examination and investigations may not detect the underlying causes many a times.[2]

Erythroderma was described by Hebra in 1868.[3] The disease is usually associated with underlying cuteneous or systemic disorder or drug intake and rarely it may be idiopathic. Erythroderma affects the skin as well as other systems of the body giving rise to haemodynamic disturbances, biochemical derangement, fever, tachycardia, hypoalbuminemia and pedal edema, in addition to various cutaneous changes.

Treatment addresses the underlying etiology, symptomatic relief and potential systemic complications.[4] Although its a rare disease, the mortality rates are low and morbidity related to it is considerably high as it is often a chronic disease with debilitating signs and symptoms such as intense pruritis and scaling. Thus it is importance to find the etiology with the special emphasis on histopathology allowing early and appropriate intervention for each case.[2]

The present study of 65 cases of erythroderma, carried out in the Department of Dermatology, Rajiv Gandhi Government General Hospital, is aimed at studying the etiopathology, clinical features, its course, evolution and associated systemic derangement.

REVIEW OF LITERATURE

ERYTHRODERMA

DEFINITION

Erythroderma is a reaction pattern characterised by generalised and confluent erythema and desquamation affecting more than 90% of the body surface area and is usually accompanied by lymphadenopathy and fever.[5,6]

Exfoliative dermatitis, also referred to as erythroderma, is an inflammatory disorder in which erythema and scaling occur in more or less genaralized distribution. [4]

Various synonyms for exfoliative dermatitis or erythroderma have appeared in the literature[2,7] They are;

1. Dermatitis exfoliativa
2. Pityriasis rubra (Hebra)
3. Erythroderma of Wilson Brock type
4. Erythroderma-red skin (Homme d Rouge)
5. Erythema scarlatiniform
6. Epidemic form of exfoliative dermatitis
7. Generalised exfoliative dermatitis
8. Red man syndrome

HISTORICAL ASPECTS

In 1868 Von Hebra described this progressive skin disorder as Pityriasis Rubra of Hebra and Wilson described it as Wilson Brock disease. Later Wilson suggested the name Dermatitis Exfoliativa in 1870. Erythema Scarlatiniform is the name suggested by Ferol in1876. In 1891 Savil described it as an epidemic form of Exfoliative dermatitis and a self limiting disorder associated with infectious agent or drug.[2] The term erythroderma was introduced by Von Hebra to describe an exfoliative dermatitis involving more than 90% of the skin surface. Based upon the clinical course, erythroderma was classified into chronically relapsing (Wilson–Brocq), chronically persisting (Hebra), and self-limiting epidemic (Savill) variants. Although of historic interest, these subdivisions are no longer employed.[8]

ETIOLOGY

Erythroderma is more common in elderly males and precipitating factors vary according to age. Males are affected between two to three times more frequently than females.[9] Erythroderma can be caused by a wide range of cutaneous and systemic diseases. As knowledge and diagnostic methods improve, the number of cases of erythroderma with an unidentifiable cause should decrease.[10]

The disease process reported to be associated with erythroderma can be classified under the following headings. [6,10-23]

1. Dermatosis
2. Systemic causes
3. Malignancy
4. Infections
5. Drugs

1. DERMATOSIS

- PAPULOSQUAMOUS DISORDERS

  Psoriasis — Lichen planus

  Pityriasis rubra pilaris — Impetigo herpitiformis

- SPONGIOTIC DERMATITIS

  Atopic dermatitis — Contact dermatitis

  Seborrhic dermatitis — Stasis dermatitis

- BULLOUS DERMATOSIS

  Pemphigus foliaceus — Bullous pemphigoid

  Paraneoplastic pemphigus — Hailey-Hailey disease

- PHOTOSENSITIVE DERMOTOSIS

  Chronic actinic dermatosis

  Actinic reticuloid

- MISCELLANEOUS

  Radiation recall dermatitis

  Ichthyosis

  Erythema gyratum repens

  Perforating folliculitis

  Pseudolymphoma

  Histiocytosis

  Mastocytosis

  Rosai-Dorfman disease

2. SYSTEMIC CAUSES

- COLLAGEN VASCULAR DISORDERS

  Subacute cutaneous lupus erythematosis

  Lupus erythematosis

  Dermatomyositis

  Reiter's syndrome

- ENDOCRINE DISORDERS

  Thyrotoxicosis

  Hypercalcitonemia

- OTHERS

  Acute graft vs host disease

  Sarcoidosis

Postoperative transfusion induced

Idiopathic hypereosinophilic syndrome

3. MALIGNANCY

- LYMPHOPROLIFERATIVE

Cuteneous T cell lymphoma

Sezary syndrome

B cell lymphoma

Angioimmunoblastic T cell lymphoma

Papuloerythroderma of Ofuji

Hodgkin's lymphoma

Castleman disease

Cutaneous anaplastic large cell lymphoma

Adult T cell leukemia

Acute myeloid leukemia

Myelodysplastic syndrome

Acute myelomonocytic leukemia

Multiple myeloma

Angioimmunoblastic lymphadenopathy

Reticulum cell sarcoma

Chronic eosinophilic leukemia

Cuteneous lymphoid hyperplasia

- SOLID TUMORS

  Lung

  Liver

  Thyroid

  Esophagus

  Prostate

  Gall bladder

  Stomach

  Rectum

  Cervix

  Ovary

  Breast

  Fallopian tube

  Melanoma

  Buschke- Loewenstein tumor

4. INFECTIONS

- FUNGAL

  Dermatophyte

  Congenital cutaneous candidiasis

  Histoplasmosis

- BACTERIAL

  Tuberculosis

  Congenital syphilis

- PARASITE

  Norwegian scabies

  Leishmaniasis

  Toxoplasmosis

- VIRAL

  Hepatitis C

  HIV

  Human herpes virus

- TOXIN MEDIATED INFECTIONS

  Staphylococcal scalded skin syndrome

  Toxic shock syndrome

5. DRUG REACTION

- ANTIBIOTIC

  Doxycycline Gentamicin

  Sulfonamides Rifampin

  Penicillins Minocycline

  Cephalosporins Neomycin

Tetracyclines	Quinidine
Streptomycin sulfate	Vancomycin
Isoniazid	Sulfasalazine
Quinine & Chloroquin	Teicoplanin
Aztreonam	Ribostamycin

- ANTIVIRAL

  Indinavir

  Zidovudine

  Dideoxyinosine

  Interferon alfa

- ANTILEPROMATOUS

  Dapsone

  Clofazimine

- ANTIFUNGAL

  Terbinafine

- ANTICONVULSANTS

Barbiturates	Carbamazepine
Phenytoin sodium	Phenobarbital

Lamotrigine

- CARDIAC DRUGS

Quinidine	Diltiazem
Practolol	Isosorbide dinitrate
Amiodarone	Nifedipine
Captopril	Thiazide
Mexiletine	

- HEAVY METALS

Arsenic	Gold
Mercury agents	Bismuth

- ANALGESICS

Aspirin	Piroxicam
Fenoprofen	Flurbiprofen
Diclofenac	Celecoxib

- DIABETICS

Sulfonylureas	Chlorpropamide

- CHEMOTHERAPHY

5-Fluorouracil	Imatinib
Vinca alkaloids	Cisplatin
Doxorubicin	Carboplatin
Mitomycin C	Pentostatin

- ANTIPSYCHIATRIC

Barbiturates	Chlorpromazine
Phenothiazines	Bupropion
Lithium	

- MISCELLANEOUS

Antimalarials	Cimetidine
Iodides	Codeine
Epoprostenol	Ephedrine
Allopurinol	Erythropoietin
Potassium thiocyanate	Interleukin - 2
Diethyl stilbestrol	Omeprazole
Vitamin A	Ranitidine
Leflunomide	Retinoids
Chlorpromazine	Thalidomide
Tumor necrosis factor- Alpha	Tramadol

In T Hasan, CT Jansen studied 50 cases of erythroderma of which 21 had pre existing dermatoses as a causative factor, 6 had topical sensitization to drugs, 5 had reactions to internal drugs, 2 had mycosis fungoidosis and 16 had idiopathic erythroderma. [24]

Yuan XY et al studied 82 patients of erythroderma from Jan 2003 to Dec 2008, in China. According to this study the most common causative factors were pre existing dermatoses (72%) followed by drug reactions (17%), idiopathic (6.1%) and malignancies (4.9%). Among the pre existing dermatoses, psoriasis is the most common cause (30.5%).[25]

Akhyani had studied 97 cases, of which the most common causative factors were pre existing dermatoses (59.7%) followed by drug reactions (21.6%), malignancies (11.3%) and idiopathic (7.2%). Carbamazepine was the most common drug (57.1%). [26]

Pal S, Haroon TS, et al had studied 90 cases, of which the most common causative factors were pre existing dermatoses (74.4%) followed by idiopathic (14.6%),drug reactions and malignancies each accounting for 5.5% . [3]

PR Bharatiya et al had studied 46 cases, of which the most common causative factors were pre existing dermatoses (67.4%) followed by drug reactions (21.74%), idiopathic (6.5%) and malignancies (4.35%).[27]

In various other studies done previously have also showed pre existing dermatoses as the most common underlying cause.[3,7,28-34]

Identifying the etiology in every case of exfoliative dermatitis is not always possible. The correlation between the clinical presentation and the etiology in exfoliative dermatitis is poor, due to the fact that changes specific to dermatitis or drug reactions are frequently masked by nonspecific changes induced by exfoliative dermatitis. A conclusive clinical-histological correlation thus may demand several biopsies. In different international studies, the rates of final etiological diagnosis based on histopathology varied from 15% to 43% of cases submitted for biopsy.[2,11]

Even after exhaustive investigations, there were cases in which the etiology of the erythroderma remained uncertain. In cases with undetermined cause, strict clinical and histological follow up are mandatory, due to the possibility of omission of drug intake or slow progression of cutaneous lymphoma. [11]

EPIDEMIOLOGY

In general, the studies have shown a male predominance, with male to female ratio approximately 1.85:1 to 4:1. The average age of disease onset among the patients varies from 41 to 61 years.[3,9,10,11,15,35-38]

Hansen and Jansen reported a incidence of 1 to 2 / 100,000.[24] Mean duration of the illness was 1.44 years in a study[39] and in a another study it was 11.81 years.[31]

PATHOGENESIS

The pathogenesis of erythroderma/exfoliative dermatitis is unclear. Currently, it is believed that the condition is secondary to an intricate interaction of cytokines and cellular adhesion molecules, including interleukins-1, 2, and 8, intercellular adhesion molecule-1 (ICAM-1) and tumor necrosis factor (TNF).[10]

These interactions result in a dramatic increase in the epidermal turnover rate, causing a higher than normal mitotic rate and an increase in the absolute number of germinative skin cells. Furthermore, the time required for cells to mature and travel through the epidermis is decreased, and is manifested as an increased loss of epidermal material, together with a significant loss of protein and folate.[40]

Consequently, scales consist of material normally retained by the skin (nucleic acids, amino acids, soluble protein), and the daily loss of scales increases from 500-1000 mg to 20-30gm[41,42]. Despite this, the lost material usually has marginal metabolic significance. Abel et al [43]studied the immunophenotypic characteristics of benign (psoriasis, dermatitis,

drug-induced) and malignant (Sezary syndrome, mycosis fungoides) forms of erythroderma, and found them to be similar. In immunohistochemical studies conducted by Sigurdsson et al[44,45] the dermal infiltrate in patients with Sezary syndrome mainly showed a T-helper-2 cytokine profile, while benign reactive erythroderma showed a T-helper-1 cytokine profile, indicating that, although clinically similar, they have different underlying pathogenic mechanisms.

CLINICAL FEATURES

Onset of erythroderma may be acute or insidious. It is characterized by universal erythema and scaling involving more than 90% of body surface area. Initially the patient may present with erythematous patches which then progress to form generalised erythema.

About 2 to 6 days after the onset of erythema, scaling begins classically over the flexures initially which then generalise to involve other body sites. The skin becomes dull red as scaling progresses.[46] In acute process scales are large with a reddish hue and in chronic cases they become small with dusky colour.[47, 48] When the scaling is universal, the skin is bright red, hot and dry. The intensity of erythema may fluctuate over a period of few days. The skin is relatively dry and there may be areas of crusting with moist lesions due to secondary infections.

In chronic cases edema and lichenification may be present causing skin induration which may in turn lead to a sensation of tightness. Chronic periorbital skin involvement may cause ectropion and epiphora.

Palmoplantar keratoderma is common in erythroderma of chronic duration, PRP, Norwegian scabies, idiopathic erythroderma and in CTCL.[49,50] Pigmentary disturbances and eruptive seborrheic keratosis may develop and as erythroderma subsides the keratosis may subside spontaneously.[50]

Other features like thermoregulatory disturbances, pruritus, malaise, fatigue are reported by the patients commonly but these symptoms are not specific to any cause.

HAIR CHANGES

Alopecia is seen in about 25% of patients. When the exfoliation has been present for some time, the scalp and body hair may be lost. Hair loss in chronic exfoliative dermatitis may be related to the diversion of sulphur containing amino acid to synthesize the skin protein instead of hair keratin.[51, 52]

NAIL CHANGES

Nail changes that are described in erythrodermic patients are discoloration, brittleness, dullness, paronychia, pitting, subungual hyperkeratosis, Beau's lines, shininess, onycholysis, onychauxis and splinter hemorrhages.[3, 13] Shiny nails are indirect defect as patients rub their hands on their skin to obtain relief from itching, preferring this to actual scratching as this does less damage.[53] Shelley[54] described alternating bands of nail plate discontinuity and leukonychia known as shoreline nails in drug induced erythroderma reflecting the period of drug intake. Nail changes may help to find out the underlying etiolgy. Hair loss and nail changes will restore back to normal after clearing of the skin.

MUCOUS MEMBRANE

Mucous membranes are usually spared, but may be involved in drug induced erythroderma. The types of mucousal lesions reported are oral erosion, diffuse oral pigmentation, angular cheilitis, oral candidiasis, geographic tongue, conjunctivitis, nasal erosions and crusting.[3,31]

DERMATOPATHIC LYMPHADENOPATHY (LIPOMELANOTIC RETICULOSIS)

Non specific lymphadenopathy occurs in exfoliative dermatitis which is probably due to reaction to the inflammatory process in the skin and is rarely of diagnostic or prognostic significance. However unusually prominent or asymmetrical lymph node enlargement may suggest lymphoma as a possible cause.[2,50] Commonly axillary, inguinal and cervical group of lymph nodes are enlarged which are discrete, mobile, non tender and have a firm rubbery consistency.[55] The presence of palpable enlarged lymph nodes is common in cutaneous T cell lymphoma. It is found in about one fourth of early cases, in 70% to 75% of advanced cases and in 90% patients with generalized exfoliative dermatitis that is with sezary syndrome.[56]

The pathogenesis of the lymphadenopathy is obscure, no definite cause is established. Scratching induced by pruritus presumably brings about the liberation of melanin from the epidermal cells, which is transferred by the lymphatic vessels to the regional lymphnodes.

Biopsy study of the enlarged lymph nodes should be performed for all patients in whom the etiology of the exfoliative dermatitis cannot be determined. In the histopathological examination, the follicular pattern

of the lymphnode will be retained in early stages. The follicles show slight or moderate increase in their germinal centres surrounded by rim of lymphocytes. The most conspicuous feature is enlarged paracortical area due to presence of large number of macrophages and pale staining cells of reticulum series. Within macrophages are aggregates of melanin, lipid and rarely haemosiderin giving rise to the term 'Lipomelanic Reticulosis'. (57)

In series by pal and colleagues lymphadenopathy was present in 55.5% of cases and in all cases it was dermatopathic lymphadenopathy except in one case where it was Hodgkin's lymphoma, in a study by chaudhary and colleagues 17 lymph node biopsy was done of which 33% cases showed dermatopathic lymphadenopathy and incidence in other studies was 21.3%,(26) 33%,(15) 22.67% (19) of cases.

HEPATOMEGALY

It is most commonly seen in drug induced erythroderma. Hepatomegaly was seen in 8% of the cases in a study by Sudho and colleagues.(39) Other studies have shown incidence of 25.5%,18% 13.33%,19.14%,15% and 2.5% of the cases.(9) Fulminant hepatitis has also been reported in a few cases.

SPLENOMEGALY

It is a rare finding in erythroderma and is most commonly associated with lymphoma. Splenomegaly was demonstrated in 8 % of cases in a study by Sudho and colleagues[39], 20% and 14% of cases in another study.[28] Lymphadenopathy along with organomegaly is suggestive of either malignancy or drug hypersensitivity.[12]

GYNAECOMASTIA

It is a common finding in almost all patients with exfoliative dermatitis of at least several weeks (longer than 6 weeks). It is apparently a hormonally mediated change although the precise mechanism is still unknown. Schuster and Brown in 1962 reported gynaecomastia secondary to unexplained hyperestrogenism. It's overall significance and incidence were unknown. Hyperestrogenism is seen by increased urinary excretion of estrogens.[29, 58]

OTHER FEATURES

Some studies reported, sparing of exfoliation over nose and paranasal area, and called it the "nose sign".[59,60,61] Satish Agarwal and colleagues suggested that this may be due to more sun exposure of this area, which might have some ameliorating effect due to its presumptive

antimitotic action and frequently blowing or rubbing one's nose. Both of these may help to remove the scale.[60] Pal and colleagues suggested that it is due to decreased responsiveness of spared area.[3]

"Deck chair sign" has been described in which erythroderma spares abdominal skin folds. This sign is classic for papuloerythroderma of Ofuji, which has been reported in Japan and Europe. Elderly men are affected with flat topped erythematous papules that coalesce into erythroderma; with sparing of abdominal, axillary and inguinal folds.[62-65] This sign is also reported in atopic dermatitis, psoriasis and idiopathic erythroderma.[3]

Generally exfoliative dermatitis is viewed as an end stage process where the identifiable features of the underlying disorder are lost. This is true in many cases but continued clinical observation may reveal local changes which are characteristic of the primary cause. [12]

There are also certain clinical signs of diagnostic importance which are associated with some underlying disorders and not with others. The distinctive clinical features of the primary disease that give rise to exfoliative dermatitis are discussed.

PSORIATIC ERYTHRODERMA

Psoriatic erythroderma may represent a generalized Koebner's reaction especially when precipitated by topical treatment. Psoriatic exfoliative dermatitis evolves from two types of psoriasis; Chronic stable form of psoriasis and unstable psoriasis. Chronic stable form of psoriasis can gradually evolve by process of koebnerisation into erythroderma. During exfoliation, psoriatic characteristics are retained and prognosis is good. In case of unstable psoriasis, the onset of erythroderma may be sudden and the characteristic feature of the disease is lost, patient is ill and itching is severe.[14, 66]

The triggering factors of psoriatic erythroderma include withdrawal of systemic steroids, excessive use of potent topical corticosteroids, abrupt discontinuation of methotrexate, topical irritants such as tars, dithranal, systemic medications such as antimalarials, lithium, terbinafine, phototherapy burns, infections including HIV, pregnancy, emotional stress, hypocalcemia and systemic illness.[11,12,66] Classic plaques of psoriasis vulgaris may be evident in early and remitting stages of erythroderma. Psoriatic arthritis and psoriatic nail changes may be present in some cases. Some times the typical nail changes of psoriasis

persist but mostly there will be nail dystrophy which occurs in exfoliative dermatitis.[12]

The various diagnostic clue to diagnose psoriasis as the underlying cause include history of silvery scaly plaques in areas like elbow, knee, scalp, lumbosacral area etc, nail changes like pits, subungual hyperkeratosis, onycholysis, oil drop sign etc, association with psoriatic arthritis and presence of collarette of scales suggestive of rupture of pustules in case of pustular psoriasis.

ECZEMA

ATOPIC DERMATITIS

Past history of atopy and characteristic distribution pattern are suggestive of the disease. Generalization of atopic dermatitis usually follows exacerbation of chronic localized eczema of the anticubital, popliteal fossae, face and neck. Pruritus is often severe, white dermographism may be prominent. Atopic epicanthal fold of the lower eye lid may be prominent. In chronic or severe atopic dermatitis, atopic cataract may be apparent. Elevated level of IgE and eosinophilia is common.[67]

SEBORRHOEIC DERMATITIS

It has typical sites of predilection such as scalp, back of ears, alae nasi, midline of chest and axilla. If patient gives history of preexisisting skin lesion over the above mentioned areas seborrheic dermatitis can be suspected.

CONTACT DERMATITIS AND STASIS DERMATITIS

Various clues like distribution of original lesion, history of contactants in case of contact dermatitis and history of pre existing venous disease in case of stasis dermatitis may help in finding the cause.

It may generalise to produce erythroderma.

PHYTOPHOTODERMATITIS

In India the plant Parthenium hysterophorous belonging to compositae family has been reported to produce erythroderma. The sensitizer in this plant is sesqueterpene lactone.

PITYRIASIS RUBRA PILARIS

The classical adult type of PRP which characteristically progress in a cephalocaudal direction is the most common type which may go in for erythroderma over a period of 2-3 months. Persistence of islands of

normal skin (Nappes Claires sign) with in exfoliated areas, follicular horny papule present on dorsal aspect of the fingers, toes, knees and elbows with waxy yellow diffuse palmoplantar keratoderma are the clinical clues to an exfoliative dermatitis arising from pityriasis rubra pilaris.[47] Pal and colleagues[3] suggested that the islands of normal skin may simply reflect a less responsive skin in these particular areas.

IMMUNOBULLOUS DISORDER

In pemphigus vulgaris few bullae may be intact, moist, erosive or crusted areas may be present due to rupture of bullae. Nikolsky's sign may be positive. Tzanck smear from an intact bullae may show acantholytic cells.[12, 55] In bullous pemphigoid there will be intense pruritus, tense bulla, urticarial plaques and erosions. Nikolsky's sign will be negative and tzank smear may not show acantholytic cells. In paraneoplastic pemphigus the patient may have recalcitrant oral lesions, erythema multiforme like lesions and failure to thrive.

LICHEN PLANUS

It is an uncommon cause of erythroderma. The violaceous colour, fine reticulate scale and angulation of the papule may be still present. Oral, penile or vaginal mucosal lesions may reveal the characteristic lacy white network pattern.[14,55]

NORWEGIAN SCABIES

This condition has been commonly seen in senile and mentally retarded patients, patients with poor cutaneous sensation (like leprosy, syringomyelia and tabes dorsalis), and patients with severe systemic disease (like leukaemias and diabetes) and in immunosuppressed patients.[68]

Clinically itching may be minimal or absent with marked crusting of hand and feet, subungual horny debris, erythematous scaly plaques occurring on neck, scalp and trunk. This may become generalized. There will be associated lymphadenopathy and eosinophilia. Mites can be easily demonstrated under microscopy on examination of the scales or crust with KOH preparation.[14,69]

DERMATOPHYTOSIS

Erythroderma has rarely been produced by chronic infection with Trichophyton violaceum.[70] Dissemination of mycotic process may be favoured by prolonged use of steroids, advancing age and focal infection.

DRUGS

A significant proportion of cases of erythroderma are due to drugs. Patients are frequently on a plethora of drugs making delineation of

etiological agent difficult. It should be remembered that topical drugs may also cause contact dermatitis related erythroderma.[9] Patients with erythroderma secondary to drug hypersensitivity syndrome appear toxic and develop systemic manifestations such as fever, leukocytosis with eosinophilia, edema, lymphadenopathy, organomegaly, liver dysfunction and renal dysfunction. Onset is usually rapid and resolution is faster when compared to erythroderma due to other causes. Exception to this is hypersensitivity reactions to drugs like allopurinol, anticonvulsants and antibiotics and this usually develops 2 to 5 weeks after starting drug therapy and may persist for weeks despite discontinuation of the drug.[7,12] Time of onset is variable as with gold and allopurinol in which case the eruption may occur after months or years. There are no special clinical signs to indicate that drugs are the cause of exfoliative dermatitis. But the ingestion of suspected drugs prior to the onset may be helpful. Usually it is of acute onset with fever and erythema which may appear first in the flexures then spread diffusely. Rash is of generalized eczema or scarlatiniform or morbilliform erythema, symmetrical in distribution associated with pruritus and oedema.[71] Oral lesions are common.[11,14,72] Morar[73] identified adverse drug reactions to antituberculosis medication as the most common cause of erythroderma in HIV-seropositive South

African patients. The list of drugs causing erythroderma is ever expanding hence it is important to consider all drug exposures.[50,74]

DAPSONE SYNDROME

Dapsone syndrome usually occurs four to five weeks after initiation of therapy. It is a hypersensitivity reaction and is not dose related.[75] The patient will present with erythroderma, generalised lymphadenopathy, hepatosplenomegaly, fever and methhaemoglobulinemia. They recover with omission of the drug and treatment with topical emollient and systemic steroids.[76,77,78]

ICHTHYOSIS

Autosomal recessive congenital ichthyosis (ARCI) includes several severe subtypes including harlequin ichthyosis (HI), lamellar ichthyosis and non-bullous congenital ichthyosiform erythroderma. Patients with these severe types of ichthyosis frequently show severe hyperkeratosis and scales over a large part of the body surface form birth and their quality of life is often severely affected. Recently, research into the pathomechanisms of these severe congenital ichthyosis have advanced dramatically and led to the identification of several causative genes and molecules underlying the genetic defects. The causative genes are

transglutaminase 1 gene (TGM1), ABCA12, two lipoxygenase genes, ALOXE3 and ALOX12B and ichthyin. [79,80]

SEZARY SYNDROME

Sezary syndrome is regarded as an erythrodermic, leukaemic variant of cutaneous T-cell lymphoma. It is characterized by generalized exfoliative dermatitis with intense itching, peripheral lymphadenopathy, and the presence of sezary cells in the cellular infiltrate of the skin and in the peripheral blood.[56]

The pre-sezary condition is defined as a chronic, steroid unresponsive erythroderma, lymphadenopathy, lymphocytic band at dermoepidermal junction and fewer than 1000 circulating sezary cells per cu mm.[81] Sezary syndrome is characterised by generalised erythroderma, associated with severe pruritus, palmoplantar keratoderma, lymphadenopathy, organomegaly and circulating sezary cells greater than 1000/cu mm. In pre-sezary, there is no abdominal organomegaly and lymphnodes reveal dermatopathic lymphadenopathy whereas in Sezary abdominal organomegaly is present and cortical and paracortical lymphnodes are infiltrated by variable number of Sezary cells along with abnormal lymphocytes.[82,83] Palmoplantar keratoderma, alopecia and onychodystrophy are progressive in Sezary syndrome. Low intermittent

levels of circulating sezary cells is also seen in cases of extensive eczema, psoriasis, atopic dermatitis and other inflammatory dermatitis.[84]

The dermal infiltrate in patients with Sezary syndrome mainly shows a T- helper 2 (Th2) cytokine profile, this in contrast to T-helper 1 (Th1) cytokine profile in benign reactive erythroderma. This indicates that although a relative uniform clinical picture of erythroderma is obvious, different pathomechanisms may be underlying.[44]

ERYTHRODERMIC CUTANEOUS T-CELL LYMPHOMA

It may occur in many ways (a) By coalescence of localized CTCL lesions to universal exfoliative dermatitis, (b) By allergic reaction as contact dermatitis or drug induced exfoliative dermatitis superimposed on CTCL, (c) By irritation with therapy or ultraviolet light. Such erythrodermic T cell lymphoma have a poorer prognosis than localized or generalised plaque T cell lymphoma.[81,85]

Patients with erythrodermic cutaneous T-cell lymphoma often have markedly depressed levels of normal blood T cells, to the range seen in advanced acquired immunodeficiency syndrome.[85]

LYMPHOMA AND LEUKAEMIA

Pruritus is often severe and exfoliation is universal with infiltration of skin and when it is severe that it can lead to leonine facies. Lichenification is a common feature. Small rounded or annular patches of skin may be spared which is an inconsistent but valuable diagnostic sign.[14,55] Large asymmetrical lymph node enlargement with specific or non specific histological changes, hepatosplenomegaly and raised ESR may be the associated features.[2] Clues for internal malignancy causing exfoliative dermatitis are

- Insidious development of exfoliative dermatitis.
- Progressive debility.
- Resistance of exfoliative dermatitis to the standard treatment
- Absence of prior skin disease. [86]

IDIOPATHIC VARIETY / "THE RED MAN SYNDROME"

The percentage of cases of idiopathic variety can be decreased with detailed examination, observation and laboratory investigations, but in any series of cases it is rarely below 10%. The cutaneous finding may precede any other evidence of lymphoma by many months or years. If those case are excluded the hard core of chronic exfoliative dermatitis of idiopathic variety consists mainly of elderly men, in whom the condition

runs a very long course with partial and temporary remissions. Pedersen and colleagues described patients with exfoliative dermatitis in whom the underlying cause could not be established as Redman syndrome, which is associated with palmoplantar keratoderma, dermatopathic lymphadenopathy, raised of serum IgE, non-specific skin histology or some pleomorphic infiltration and exfoliative dermatitis extending over one month.[14,87] It is occasionally referred to as "I homme rouge". The three most common cause of protracted idiopathic erythroderma are probably atopic eczema of the elderly, intake of drugs overlooked by the patient and prelymphomatous eruptions.

CAUSES OF NEONATAL AND INFANTILE ERYTHRODERMA

(88,89,90)

Infections

Staphylococcal scalded skin syndrome

Toxic shock syndrome

Generalized congenital and neonatal candidiasis

Staphylococcal pustulosis

Ichthyoses

Non-bullous ichthyosiform erythroderma

Bullous ichthyosiform erythroderma

Harlequin ichthyosis

Lamellar ichthyosis

Conradi-Hunnerman syndrome

Netherton’s syndrome

Immunodeficiency syndromes

Omenn’s syndrome

Graft versus host reaction

Hypogammaglobulinemia

Di George's syndrome

Severe combined immunodeficiency

Cutaneous T-Cell lymphoma

Metabolic disorders

Disorders of biotin metabolism

Essential fatty acid deficiency

Others

Atopic dermatitis

Infantile seborrheic dermatitis

Psoriasis

Pityriasis rubra pilaris

Diffuse mastocytosis

Protein malnutrition

Holocarboxylase deficiency

Acrodermatitis enteropathica

Leiner’s disease

Protein malnutrition

Pruszkowski A et al had studied 51 cases who presented with erythroderma during their first year of life. The underlying causes observed were immunodeficiency (30%), simple or complex ichthyosis(24%), Netherton’s syndrome (18%), eczematous or papulosquamous dermatitis(20%) and idiopathic(8%).[91]

The main complications were hypernatremic dehydration, infections and failure to thrive. Erythroderma is a potentially life threatening condition in infants.

The management of erythroderma, regardless of underlying cause includes correction of caloric and protein intake, proper balance of oral and parenteral fluids and electrolytes, and prevention and treatment of infection.[89]

ERYTHRODERMA IN HIV POSITIVE PATIENTS

The common causes of erythroderma in HIV patients are

1. Psoriasis
2. Seroconversion in HIV positive patients
3. Hypersensitivity to drugs
4. Seborrhoeic dermatitis
5. Lymphoma
6. Norwegian scabies

In a study done by Morar N et al[73] drug reaction was found to be the commonest cause of erythroderma among HIV positive patients.

HISTOPATHOLOGICAL CLUES FOR THE DIAGNOSIS OF ERYTHRODERMA[9,11,56,92]

Psoriasis

Epidermal hyperplasia, parakeratosis, neutrophils in cornified layer, diminution or disappearance of the granular layer at least focally, suprapapillary thinning, dilated and tortuous blood vessels that spiral up within dermal papillae, superficial perivascular lymphohistiocytic infiltrate and neutrophils in infiltrate.

Cutaneous T- cell lymphoma

Perivascular or lichenoid infiltrate mainly composed of lymphoid cells within papillary dermis. Presence of small or medium- sized lymphocytes with irregular or convoluted nuclei. Epidermotrophism or Pautrier's microabscess. Wiry collagen bundles in haphazard array in the papillary dermis.

Pityriasis rubra pilaris

Alternating orthokeratosis and parakeratosis (vertically and horizontally), focal or confluent hypergranulosis, thick suprapapillary plates, sparse superficial perivascular lymphohistiocytic infiltrate and dialated non tortuous capillaries in the dermis

Drug induced erythroderma

Vacuolar alteration of basal keratinocytes. Scattered necrotic keratinocytes along the basal layer and/or above it. Perivascular or lichenoid lymphohistiocytic infiltrate involving dermal-epidermal interface

Dermatitis (eczema)

Moderate or prominent suprapapillary spongiosis, parakeratosis, edema of papillary dermis and perivascular superficial lymphohistiocytic infiltrate with many eosinophils.

Pemphigus

Subcorneal bulla and acantholytic keratinocytes.

Bullous pemphigoid

Subepidermal bullae with eosinophilic infiltrate.

Actinic reticuliod

Hyperkeratosis, acanthosis and atypical mononuclear cells admixed in the superficial and deep dermal infiltrate with or without spongiosis.

Dermatophytosis

Hyphae within stratum corneum, mounds of parakeratosis.

Scabies

Scabetic mite in the stratum corneum. Perivascular and interstitial infiltrate of eosinophils.

Seborrheic dermatitis

Spongiotic psoriasiform dermatitis with parakeratosis often with neutrophils at the tips of follicular ostia.

Dermatomyositis or subacte lupus erythematosis

Interface dermatitis with vacuolar alteration, often thickening of the basement membrane, colloid bodies, and increased dermal mucin.

Sarcoidosis

Non caseating epitheloid dermal granulomas with little or no mantle of surrounding lymphocytes.

Lymphoproliferative diseases

Superficial, deep and interstitial dermal infiltrate of atypical mononuclear cells.

Source of major concern is exocytosis that characterizes the pattern of early psoriasis and basal epidermotropism of early CTCL. In such cases clues to the diagnosis are (i) the numerous lymphocytes and scanty spongiosis in CTCL whereas, in psoriasis lymphocytes are usually sparse (ii) marked edema of papillary dermis in psoriasis but usually not in CTCL, atypical lymphocytes within the dermis and epidermis in CTCL

but not in psoriasis (iii) the lichenoid arrangement of the dermal infiltrate mostly composed of lymphocytes in CTCL but not in psoriasis.[92]

COMPLICATIONS OF ERYTHRODERMA [11-14,47]

1) Fluid and electrolyte imbalance
2) Hypoalbuminemia
3) Thermoregulatory disturbance
4) High output cardiac failure
5) Capillary leak syndrome
6) Infection
7) Acute respiratory distress syndrome
8) Gynecomastia
9) Metabolic disturbance
10) Hematological disturbance
11) Dermatogenic enteropathy

Management

Hospitalisation is a must with skilled nursing care. The initial management of erythroderma is the same regardless of etiology. This should include replacement of nutritional, fluid and electrolyte losses.[12] The environmental temperature must be carefully regulated. Local skin-care measures should be employed, such as oatmeal baths as well as wet

dressings to weeping or crusted sites followed by the application of bland emollients and low-potency corticosteroids. [11] Known precipitants and irritants should be avoided and underlying cause, with its complications, are to be treated.[12,13,72] Secondary infections are treated with antibiotics. Heng[93] has suggested that colonization of skin by Staphylococcus aureus may cause erythroderma, which will clear with appropriate antibiotics. Antihistamines can be added if the patient has itching. Edema in dependent areas, such as in periorbital and pedal areas, may require diuretics.[11] Hemodynamic or metabolic instability should be treated adequately. Serum protein, electrolyte and blood urea levels should be monitored. This condition may resist therapy until the underlying cause is treated; hence it is important to determine the underlying etiology early in its management.[11,94,95]

COURSE

The disease course is greatly influenced by the etiology. It is rapidly progressive when due to drug allergy, lymphoma, leukemia, contact allergens or staphylococcal scalded skin syndrome. A slower course is observed if from a primary skin disease such as psoriasis or atopic dermatitis.[9,12,13,29] Drug-induced erythroderma patients recover completely with prompt diagnosis and treatment.[12,13] The outcome is

unpredictable in idiopathic erythroderma, and its course is marked by multiple exacerbations.[13,72]

PROGNOSIS

Prognosis of erythroderma depends on the underlying disease process. If the cause can be removed or corrected, the prognosis is generally very good. If erythroderma is the result of a generalised spread of a primary skin disorder such as psoriasis or dermatitis, it usually clears with appropriate treatment of the skin disease but may recur at any time. In case of drug induced erythroderma the duration of the disease process is short lived, resolving once the offending drug is stopped. If erythroderma is due to unknown cause the course is often unpredictable with multiple episodes of remissions and exacerbations.[11]

AIMS & OBJECTIVES

(1) To study the incidence of erythroderma in relation to age and sex.

(2) To study the etiology, clinical features & course of the disease.

(3) To study the histopathology in relevant cases.

(4) To study the metabolic and systemic complications.

MATERIALS AND METHOD

The study was conducted among individuals attending the outpatient dermatology clinic as well as the inpatients of the dermatology ward of Rajiv Gandhi Government General Hospital, Chennai during the period October 2010 - September 2012. All new cases of erythroderma of both sexes and of all age groups who attended the dermatology clinic during the said period, were taken for this study.

METHOD OF COLLECTION

A total of 65 patients of erythroderma were selected. It is a prospective observational study. The cases were analysed in detail as follows. All patients erythema and scaling involving more than 90% of the body surface area as calculated by Wallace's rule of nine were enrolled in the study in accordance with the definition of erythroderma.

A detailed history was recorded and a through clinical examination was performed for each patient. At first, preliminary data in the form of age, sex, occupation etc were noted. Then a detailed history regarding onset, evolution, duration, symptoms, previous skin disease or drug intake were noted. Any history of recurrence and precipitating factors like drugs, topical applications and infective episodes prior to onset of disease were also noted.

I. Each patient was examined and the following details were noted:

Type of scales

Sizes of scales

Color of scales

Associated lichenification / fissuring

Ectropion, eclabion

Pedal edema

Examination of scalp for scales, hair loss

Examination of nails and mucosa

Associated arthritis

Specific signs like sparing of flexures and nose sparing sign.

II. The following investigations were carried out -

Haematological investigations:

Haemoglobin percentage

Total and differential leucocyte count

Erythrocyte sedimentation rate

Peripheral smear study

Platelet count

Biochemical investigations:

Blood sugar and urea

Serum creatinine

Serum electrolyte

Serum calcium and uric acid

Total proteins, albumin globulin ratio

Other investigations:

Chest X ray

Motion for occult blood

USG abdomen

ECG

Blood VDRL

Skin biopsy

Scraping for fungus and Acarus mite, lymph node biopsy, Tzanck test, direct immunofluorescence, patch test and HIV ELISA were done in selected cases. In adults, per rectal examination was carried out to detect underlying malignancy. The course and duration of the disease was studied and episodes of recurrence were noted.

The patients who were hospitalized were given supportive treatment along with systemic steroids in deserving cases, which was withdrawn gradually and later specific treatment was instituted for the underlying dermatoses.

OBSERVATIONS & RESULTS

In the two year study period, 65 patients were diagnosed as erythroderma in our department. Various observations made are as follows.

INCIDENCE

Between the period Oct 2010 - Sep 2012, 217551 patients attended the OPD department. Among them, 65 patients were diagnosed as erythroderma and the incidence of erythroderma during the period was 0.029%

Table No.1: Incidence of erythroderma

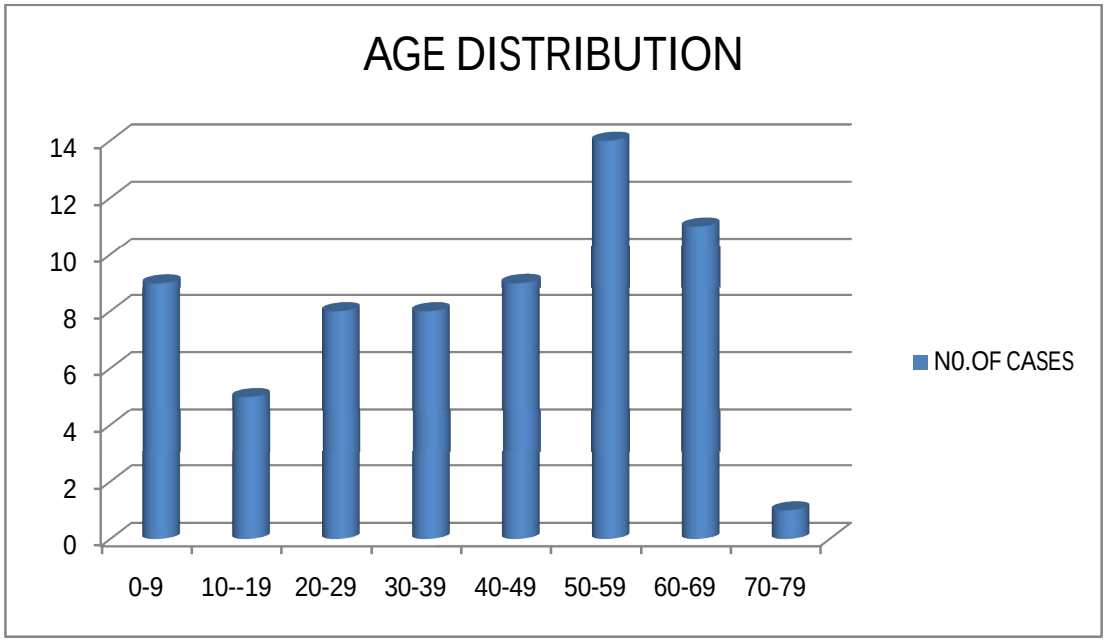
Study period	Total No.of patients	No.of cases of erythroderma	Percentage
Oct 2010 - Sep 2012	217551	65	0.029

AGE DISTRIBUTION

Age of the patients ranged from 50 days to 71 years with the average age being 40.25 years. Maximum number of patients belonged to the age group of 50-59 years (21.53%).

Table No.2: Age distribution of erythroderma

Patient's age	Number (n=65)	Percentage
0-9	9	13.84
10-19	5	7.69
20-29	8	12.30
30-39	8	12.30
40-49	9	13.84
50-59	14	21.53
60-69	11	16.92
70-79	1	1.53

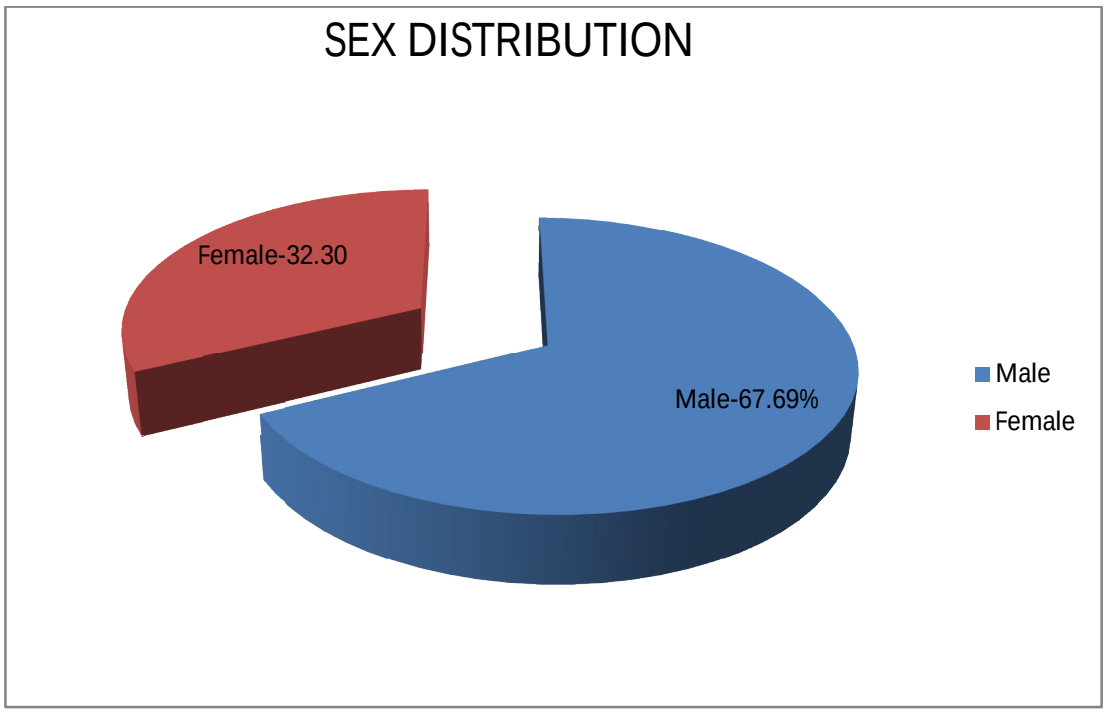


SEX DISTRIBUTION

Among 65 patients, 44 were male (67.69%) and 21 were female (32.30%), male to female ratio being 2.1:1 showing a male predominance.

Table No.3: Sex distribution of erythroderma

Sex	No.of cases (n=65)	Percentage
Male	44	67.69
Female	21	32.30

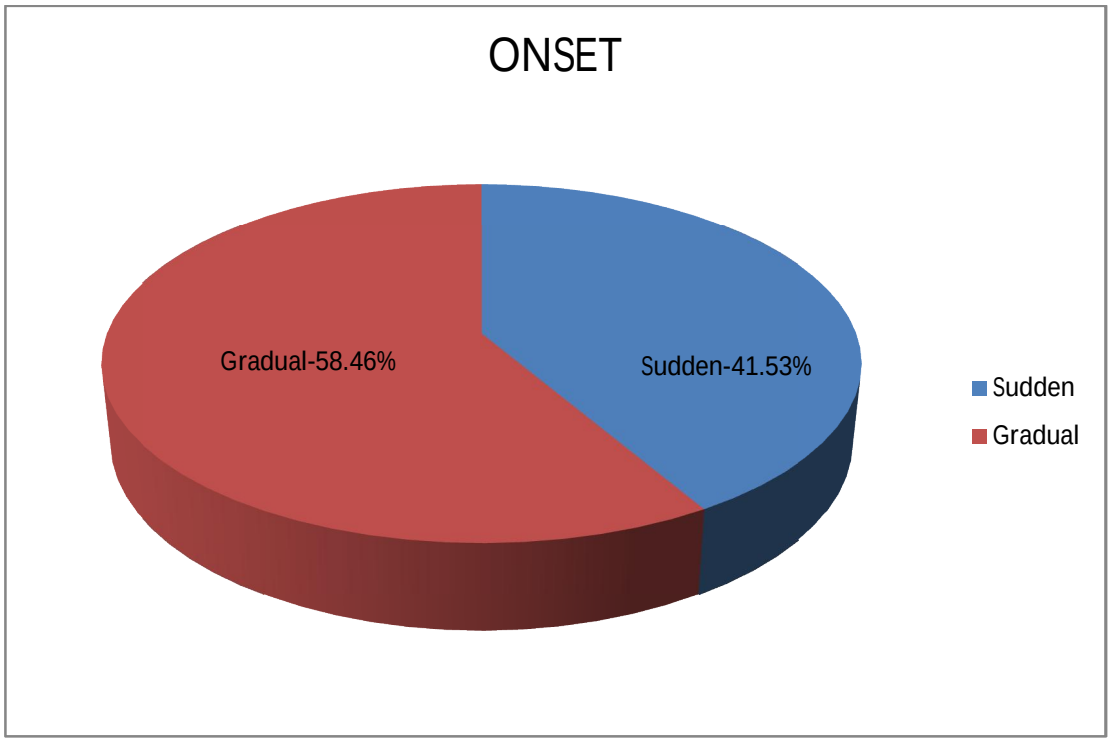


ONSET

Majority (58.46%) of patients had insidious onset of erythroderma, 41.53% of patients had sudden onset.

Table No.4: Onset of the illness

Onset	No.of cases (n=65)	Percentage
Sudden	27	41.53
Gradual	38	58.46

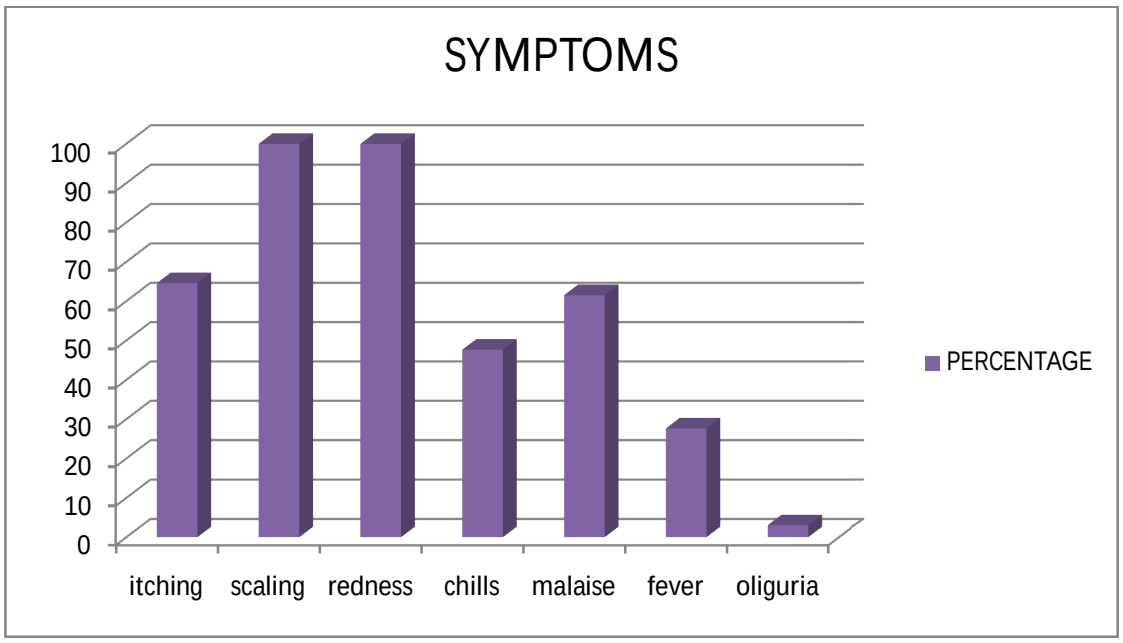


SYMPTOMS

All the patients had generalized erythema involving more than 90% of their body surface area and various degrees of scaling. Itching, the most common complaint, was recorded in 42 patients (64.62%). Fourty patients (61.54%) had complaints of malaise, thirty one patients (47.69%) had chills and eighteen patients (27.69%) had fever. Only two patients (3.07%) in the study had oliguria. None of the patients complained of loose stools suggestive of dermatogenic enteropathy.

Table No.5: Frequency of presenting complaints

Symptoms	No.of cases	Percentage
Itching	42	64.62
Scaling	65	100
Redness	65	100
Chills	31	47.69
Malaise	40	61.54
Fever	18	27.69
Oliguria	2	3.07



PHYSICAL EXAMINATION FINDING

Nail changes were seen in 45 patients (69.23%). They were commonest in cases of psoriatic erythroderma. Twenty nine patients(44.61%) had eye involvement in the form of conjunctival congestion, ectropion and icterus. Mucosal involvement was seen in 6 patients (9.23%).

Most of the patients had involvement of scalp in the form of exfoliation. Fifteen patients (23.07%) had diffuse alopecia. One patient had tinea amiantacia and 5 patients (7.69%) had psoriatic corona.

Involvement of palms and soles was seen in 26 patients (40%). Six patients (9.23%) had palmoplantar keratoderma. Among them two had pityriasis rubra pilaris, two had psoriasis, one had idiopathic

erythroderma and one had malignancy induced erythroderma. Four patients (6.15%) had instep involvement. Crusting of palms and soles was seen in a patient with Norwegian scabies.

As far as the systemic features are concerned 18 patients (27.69%) had increased temperature (>38^{o}C), 17 patients (26.15%) had pallor, 21 patients (32.30%) had lymphadenopathy, 2 patients (3.07%) had hepatosplenomegaly and 26 patients (40%) had significant pitting pedal edema.

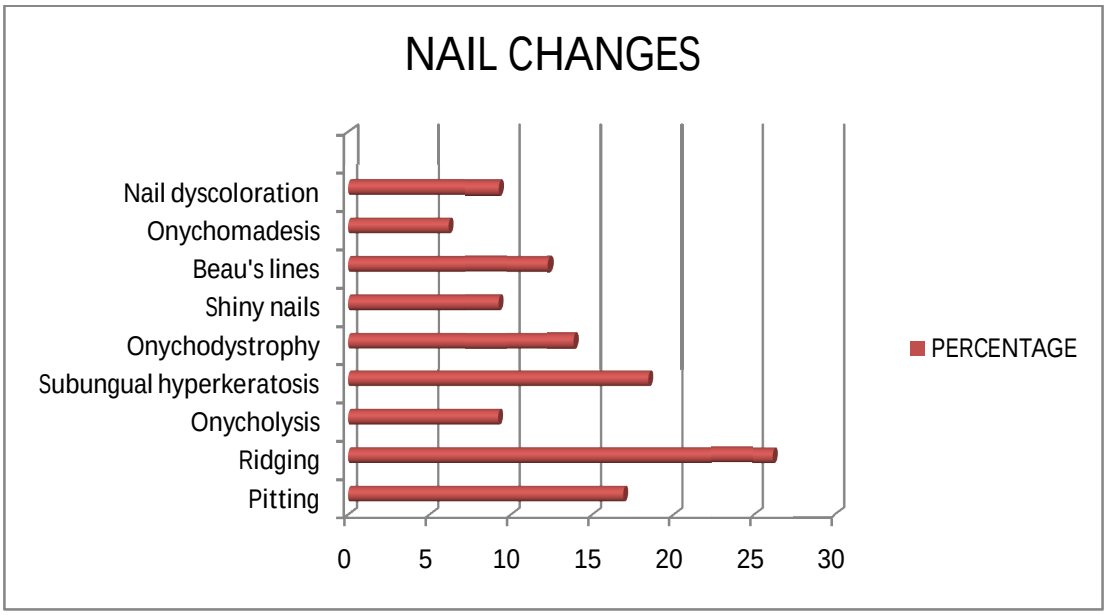
NAIL CHANGES

Forty five patients (69.23%) had nail changes. Ridging of the nail was the most common change, which was seen in 17 patients (26.15%).

Other nail findings observed were pitting (16.92%), subungual hyperkeratosis (18.46%), onychodystrophy (13.84%), Beau's lines (12.30%), onycholysis (9.23%), shiny nails (9.23%), onychomadesis (6.15%) and nail discoloration (9.23%).

Table No.6: Nail changes in erythroderma

Nail changes	No.of cases	Percentage
Pitting	11	16.92
Ridging	17	26.15
Onycholysis	6	9.23
Subungual hyperkeratosis	12	18.46
Onychodystrophy	9	13.84
Shiny nails	6	9.23
Beau's line	8	12.30
Onychomadesis	4	6.15
Nail discoloration	6	9.23

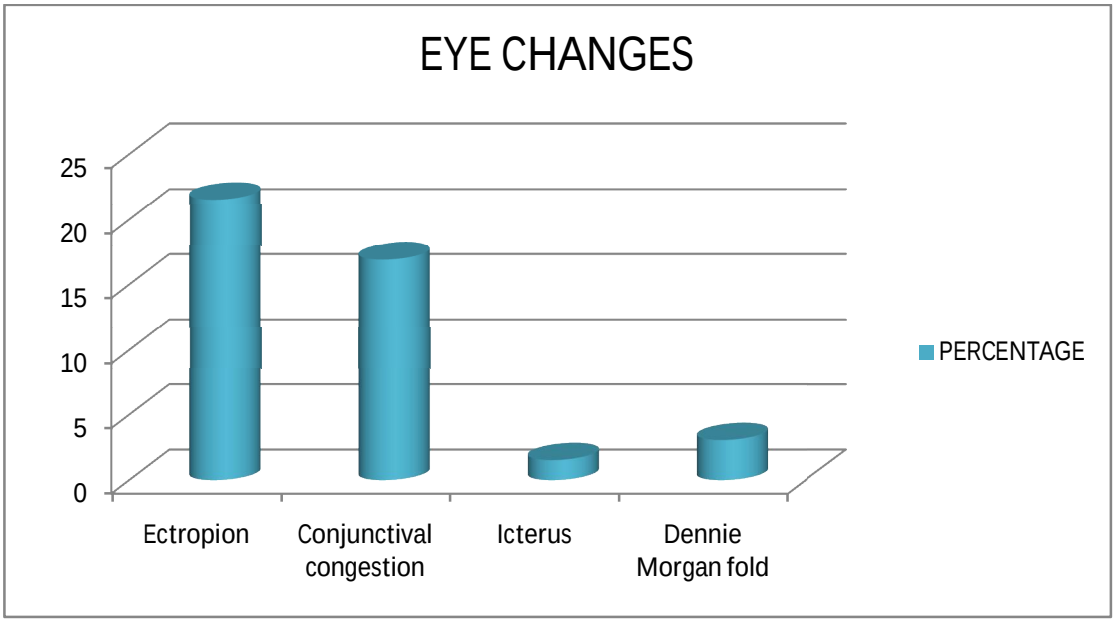


EYE INVOLVEMENT

Eye changes were seen in 29 patients (44.61%). Fourteen patients (21.54%) had ectropion among whom three had lamellar ichthyosis. Eleven patients (16.92%) had conjunctival congestion. One patient (1.53%) with Dapsone syndrome had icterus and two patients (3.07%) had Dennie Morgan fold.

Table No.7: Frequency of eye changes in erythroderma

Eye changes	No.of cases	Percentage
Ectropion	14	21.54
Conjunctival congestion	11	16.92
Icterus	1	1.53
Dennie Morgan fold	2	3.07



PEDAL EDEMA

It was seen in a 26 patients (40%). The edema was bilateral, pitting and non tender. One patient had bilateral elephantiasis due to filariasis.

LYMPHADENOPATHY

Lymphadenopathy was seen in 21 patients (32.30%). The commonest nodes affected were inguinal and axillary nodes. None of the patients had cervical lymphadenopathy. Enlarged lymphnodes were firm, mobile and discrete. One patient with non Hodgkin's lymphoma had enlarged axillary and inguinal nodes.

HEPATOSPLENOMEGALY

It was noted in 2 patients (3.07%). One patient had non Hodgkin's lymphoma and the other patient was a case of dapsone syndrome.

GYNAECOMASTIA

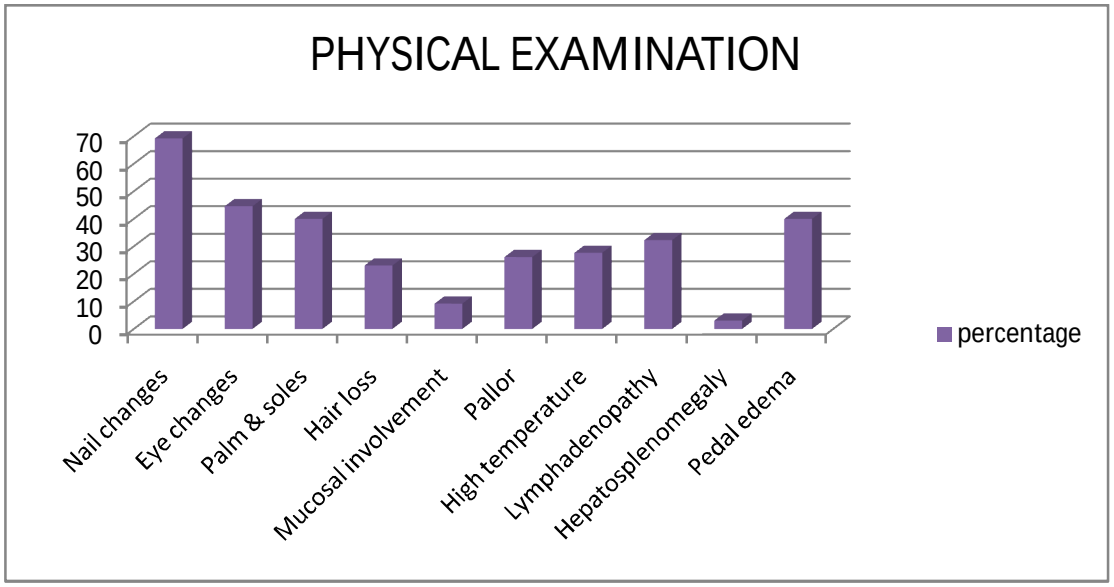
Gynaecomastia was seen in two patients (3.07%). None of them showed any underlying liver pathology or testicular atrophy. Five patients (7.69%) showed macrothelia.

Scales were grayish white or white in color and non adherent in most of the cases. Adherent moist scales and crust were seen in a patient with pemphigus foliaceous. In two patients with lamellar ichthyosis, the scales were large plate like, dark colored and adherent. Nose sign, that is sparing of nose of erythema and scaling was seen in 8 patients (12.30%).

In about 13 patients (20%), the erythema and scaling over the face was less compared to other body parts (relative sparing of face). Sparing of abdominal folds (Deck chair sign) was seen in 7 cases (10.76%). Four of them had psoriasis while three had eczema. Islands of normal skin (Nappes claires sign) was seen in two patients (3.07%). Both the patients had erythrodema with pityriasis rubra pilaris as the underlying dermatological condition. Two children with lamellar ichthyosis had eclabion and hypoplastic ears. Oral mucosal involvement in the form of congestion and erosion was seen in 6 patients (9.23%) and all the 6 cases were drug induced erythroderma.

Table No.8: Physical examination findings among the patients

Physical examination	No.of cases	Percentage
Nail changes	45	69.23
Eye changes	29	44.61
Palms & soles involvement	26	40
Hair loss	15	23.07
Mucosal involvement	6	9.23
Pallor	17	26.15
High temperature	18	27.69
Lymphadenopathy	21	32.30
Hepatosplenomegaly	2	3.07
Pedal oedema	26	40



Final diagnosis was the result of evaluation of the clinical, biochemical, histiological findings and of the evolution of erythroderma along with history given by the patients in each individual case.

ETIOLOGICAL CAUSES

Etiologically patients were categorized into four groups:

The first group comprised of pre existing dermatoses (40 patients, 61.53%): psoriasis, 20 (30.76%); eczema, 11 (16.92%); ichthyosis, 4 (6.15%); pemphigus foliaceous, 2 (3.07%); PRP, 2 (3.07%); and crusted scabies, 1 (1.53%). Among the patients with pre existing dermatoses, psoriasis was the most common cause followed by eczema.

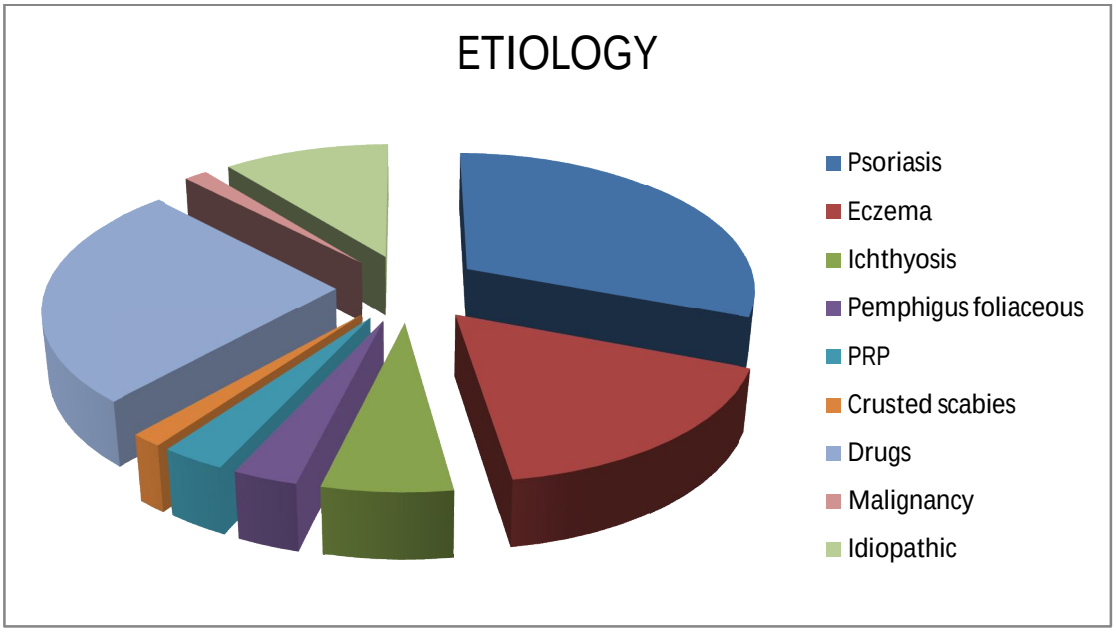
The second group, which comprised of drug induced erythroderma, had 17 (26.15%) patients. Carbamazepine was the most common etiological agent in 5 patients followed by native medicine in 3 patients, anti retroviral therapy in 2 patients, sodium valproate, antituberculous drugs, dapsone and ofloxacin in 1 patient each. In the remaining three patients the nature of the drug could not be found out. Relationship between a drug and erythroderma was established from the history of antecedent intake of the incriminating drug preceding the onset of erythroderma and clearing of the manifestations following withdrawal of the drug.

The third group comprised of malignancy associated erythroderma (1 patient, 1.53%).

The last group was idiopathic, which comprised of 7(10.76%) patients, with no specific history, associated factor or specific histology.

Table No.9: Etiology of erythroderma

Underlying cause	No.of cases (n=65)	Percentage
Pre existing dermatoses	40	61.53
Psoriasis	20	30.76
Eczema	11	16.92
Ichthyosis	4	6.15
Pemphigus foliaceous	2	3.07
PRP	2	3.07
Crusted scabies	1	1.53
Drugs	17	26.15
Malignancy	1	1.53
Idiopathic	7	10.76



LABORATORY INVESTIGATIONS

Low haemoglobin was seen in 17 patients (26.15%), increased ESR was seen in 20 patients (30.76%), hypoproteinemia was seen in 8 patients (12.30%), eosinophilia was seen in 8 patients (12.30%), atypical lymphocytes in peripheral smear was seen in one patient (1.53%) with non Hodgkin's lymphoma induced erythroderma. Five patients (7.69%) had altered liver function test and all of them had drug induced erythroderma. One patient (1.53%) had increased serum creatinine.

Tzank smear positivity was seen in two patients of pemphigus foliaceous. In a patient with crusted scabies, acarus mite was demonstrated by scrapping. Patch test with parthenium was positive in 6 patients and with potassium dichromate in 1 patient. USG abdomen

revealed hepatosplenomegaly in a case of non Hodgkin's lymphoma and Dapsone syndrome.

SKIN BIOPSY

Histopathological examination of the skin biopsy was performed in 53 patients (81.53%) under local anesthesia. Skin biopsy was not performed in some of the cases because the cause of erythroderma was clear from the start. Skin biopsy was performed in all the cases classified as malignancy and idiopathic. The biopsies were often performed within the first three days after the patients were admitted in the hospital. Among the 53 patients in whom the biopsy were done, the histopathological diagnosis was consistent with the clinical diagnosis in 23 (43.39%) patients.

In about 17 cases of psoriatic erythroderma, biopsy showed histopathological features like hyperkeratosis, parakeratosis, regular acanthosis, suprapapillary thinning and dilated dermal blood vessels with inflammatory infiltrate. Spongiform pustules of kogoj was present in about 9 cases only.

Drug reactions did not show any characteristic features except in one patient whose skin biopsy showed eosinophilic infiltrate in the

dermis. In case of the pemphigus foliaceous and PRP histopathology showed characteristic features of the disease.

In case of non Hodgkin's lymphoma skin biopsy showed non specific inflammatory infiltrate in upper dermis.

LYMPH NODE BIOPSY

Out of the three lymph node biopsies performed, one revealed features of non Hodgkin's lymphoma. The other two cases showed retained follicular architecture, enlarged paracortical area and melanophages in plenty suggestive of dermatopathic lymphadenopathy.

COURSE

The group associated with best prognosis was that related to drugs, who improved dramatically after stopping of the offending drug along with supportive measures. Patients with psoriatic erythroderma responded well with complete resolution of erythema and scaling. The other patients with pemphigus foliaceous, crusted scabies and PRP also respond well with therapy. Patients with lymphoma and ichthyosis did not show good response with treatment.

Recurrence was noted among 9 patients (13.84%) during the study period of which 3 were erythroderma of unknown etiology and 6 were psoriatic erythroderma. During the study period two patients with erythroderma died. One was due to non Hodgkins lymphoma and the other was a case of pustular psoriasis.

FIG 1. A case of psoriatic erythroderma with relative sparing of face

FIG 2- Chronic plaque type psoriasis going in for erythroderma

(A)

(B)

FIG 3 A & B – Erythema, scaling and pustules in a case of pustular psoriasis

FIG 4 – Psoriatic erythroderma with pityriasis amiantecea

(A)

(B)

FIG 5: (A) & (B) Crusting and superficial erosions in pemphigus foliaceous

FIG6: PRP showing follicular papules and islands of normal skin

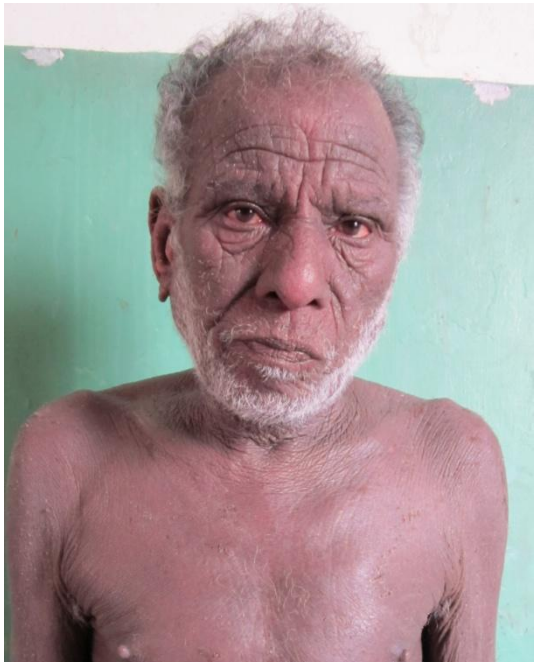


FIG 7: Phytophotodermatitis with Denny morgan folds

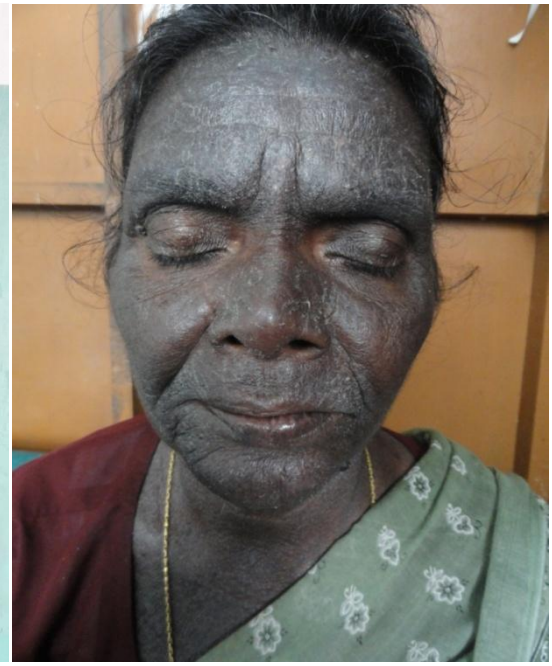


FIG 8 : Air born contact dermatitis

(A) **(B)**

(C)

FIG 9 : Lamellar ichthyosis – (A&B) showing ectropion, eclabion (C) showing plate like scales

FIG 10: Dapsone syndrome with icterus

FIG 11: Drug induced erythroderma

FIG 12: Nail changes

Twenty nail dystrophy

Onycholysis & Leuconychia

Nail discoloration

Beau’s line

Onychomadesis

Pitting

Subungual hyperkeratosis

(A)

(B) PRP sandal – yellow waxy keratoderma

(C) Palmoplantar keratoderma with fissures

FIG 13: (A), (B) & (C) Palmoplantar keratoderma

FIG 14: Instep involvement in psoriasis

FIG 15: Peeling of skin over the soles in drug induced erythroderma

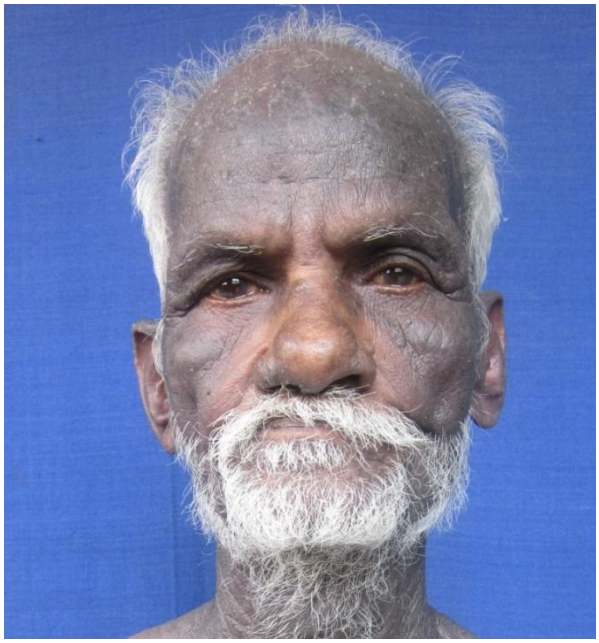


FIG 16: Nose sparing sign

FIG 17: Sparing of abdominal folds

FIG 18: Macrothelia

FIG 19: Geographic tongue

FIG 20: Conjunctival congestion

FIG 21: Bilateral pedal edema

(A)

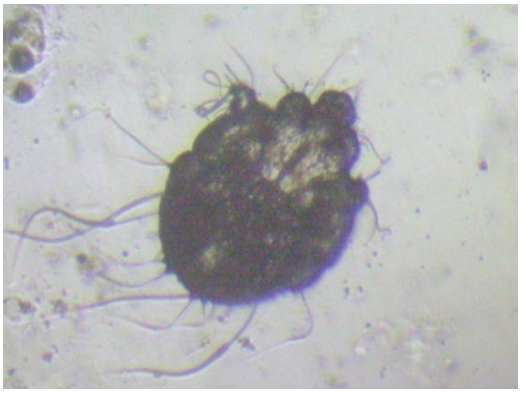
(B)

FIG 22 (A) A case of psoriatic erthroderma with diffuse hair loss
(B) Same patient with complete regrowth of hair after recovery

(A)

(B)

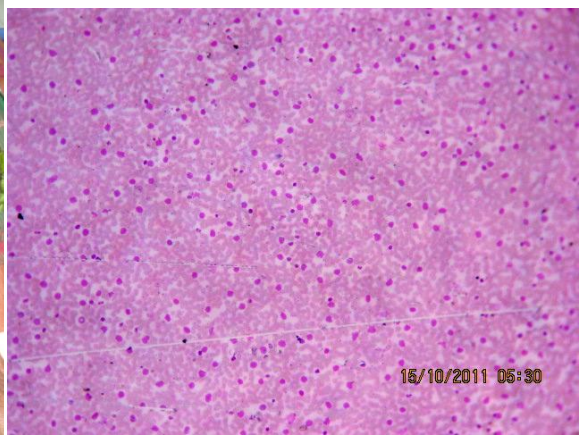
(C)



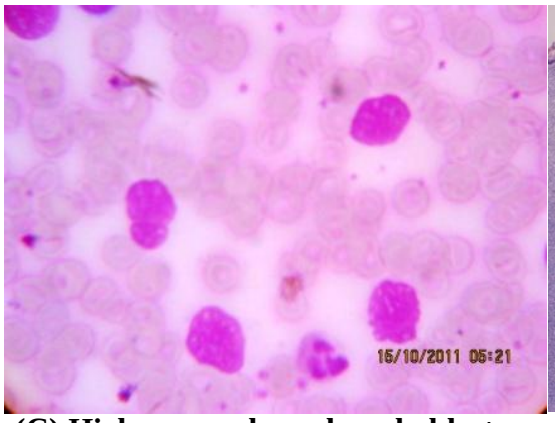
(D)

FIG 23: (A), (B), (C) & (D) Norwegian scabies

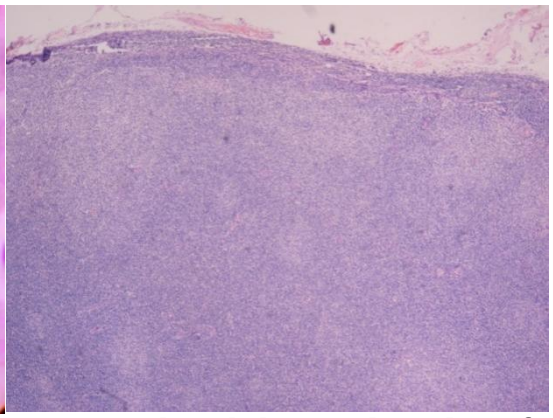
(A)



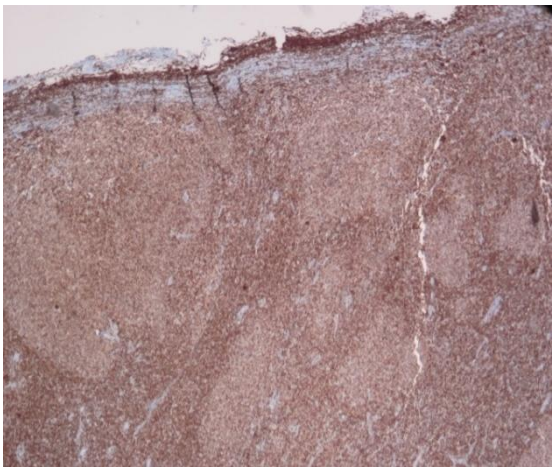
(B) Peripheral smear shows leucocytosis



(C) High power shows lymphoblast



(D) Lymph node biopsy shows loss of architecture and infiltrated with atypical lymphoid cells



(E) Immunohistochemical stain positivity with CD-20

FIG 24: (A), (B), (C), (D) & (E) Non Hodgkin's lymphoma

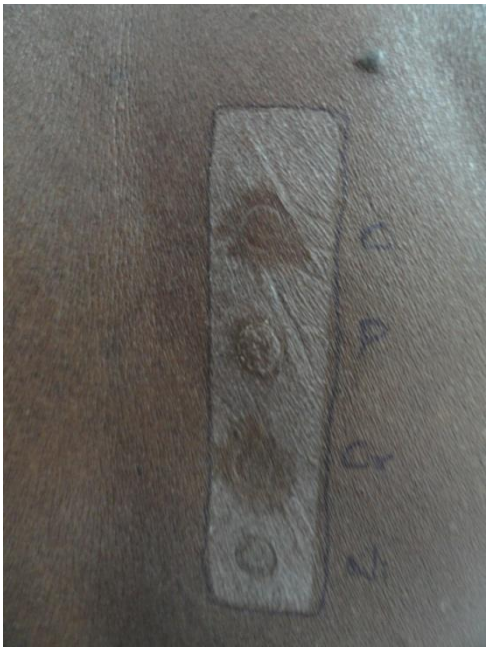
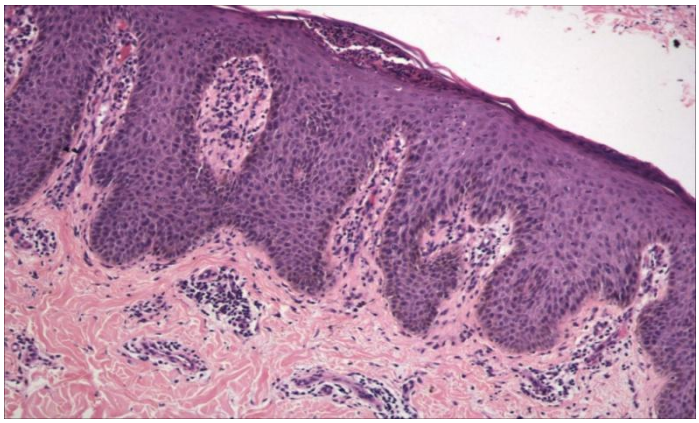
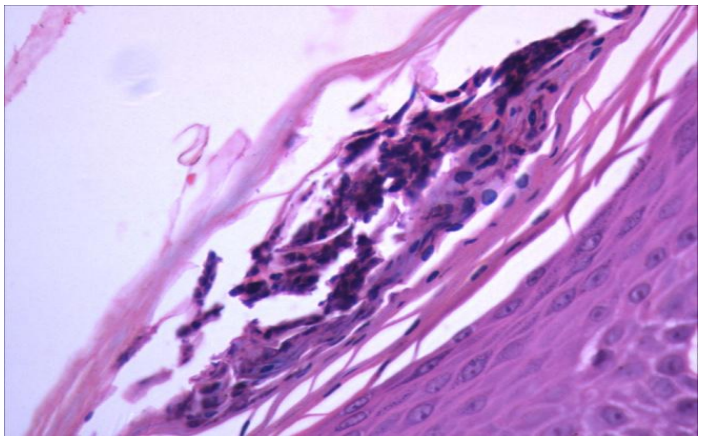


FIG 25: Patch test positive for parthenium

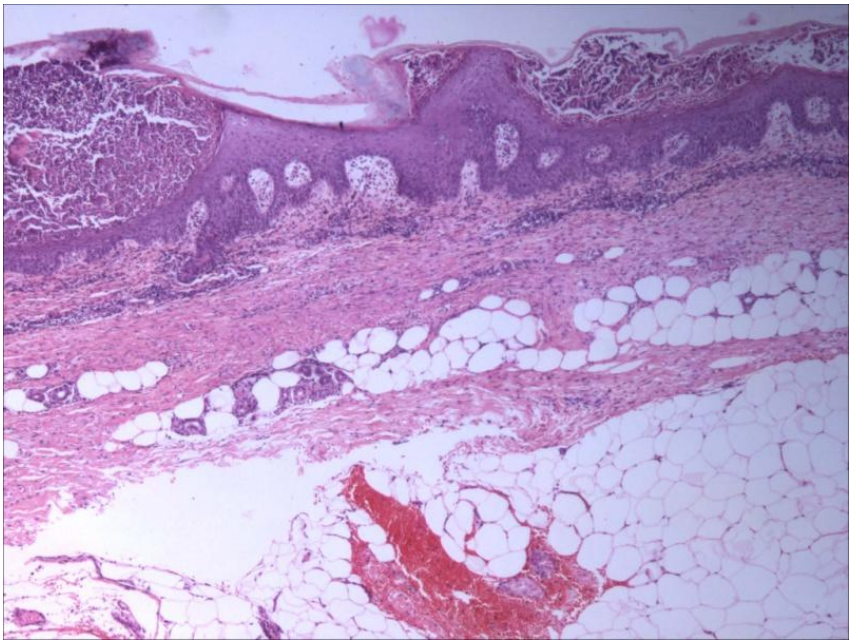


(A)

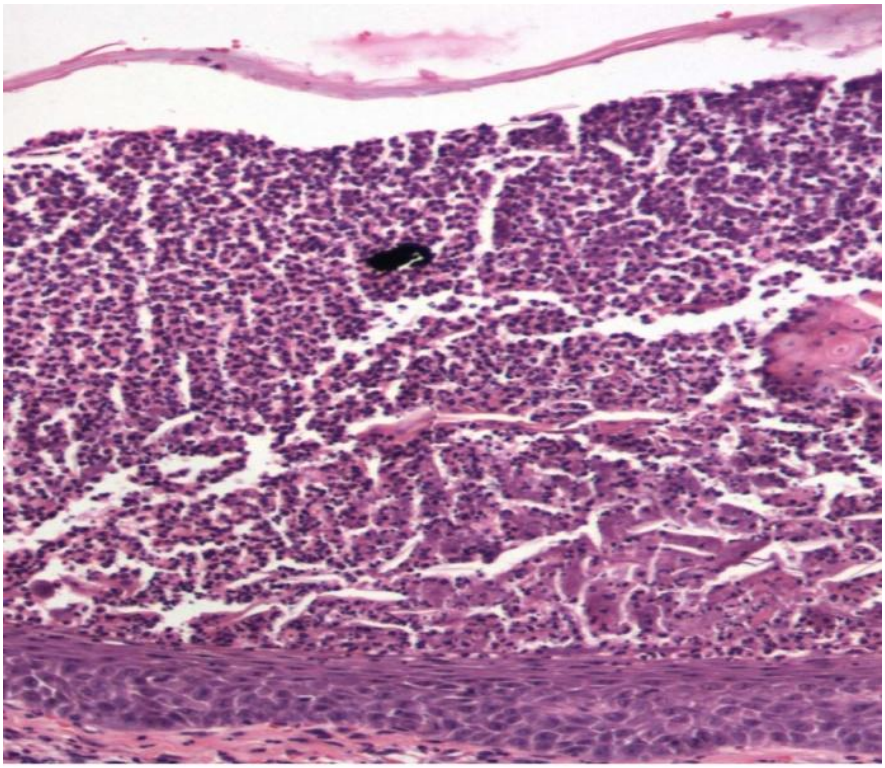


(B)

FIG 26 (A & B): Psoriasis. Regular acnthosis and munro microabscess

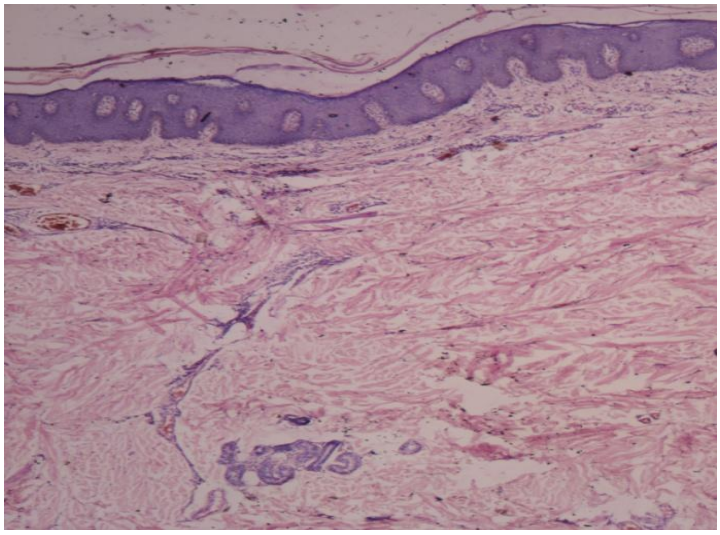


(A)

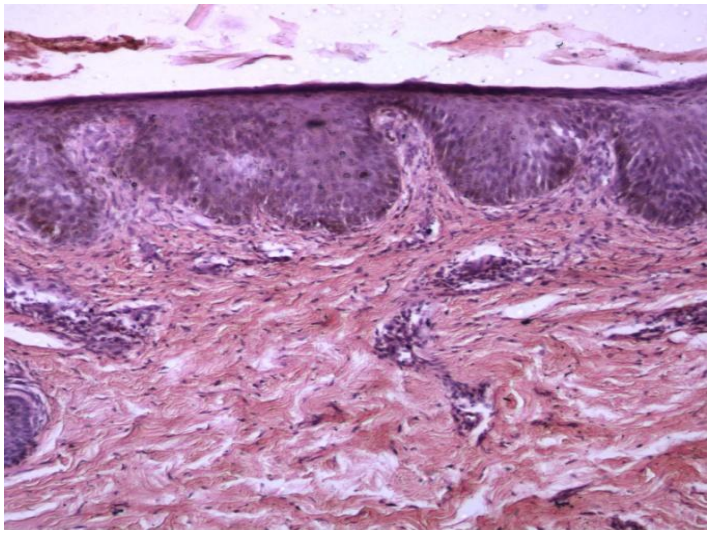


(B)

FIG 27(A & B): Pustular psoriasis. Large collections of neutrophils with spongiosis in granular and upper spinous layer



(A)



(B)

FIG 28 (A) & (B) : Non specific dermatitis. Mild lymphocytic infiltration seen in dermis

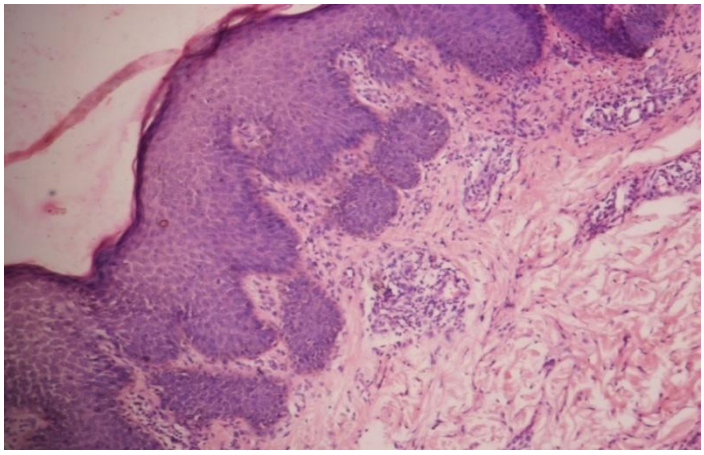
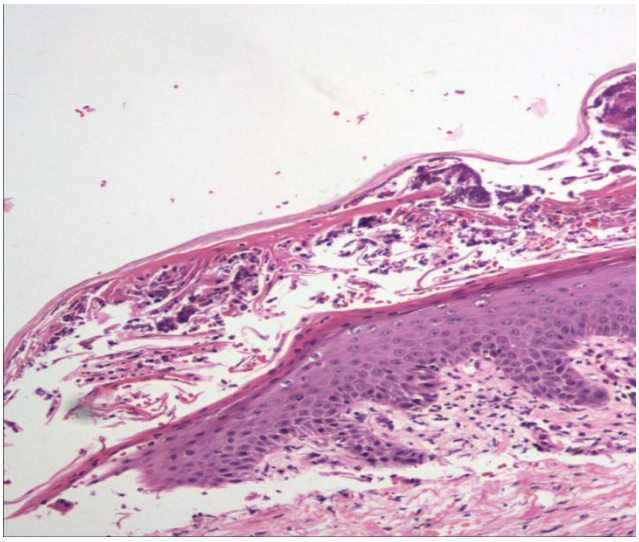
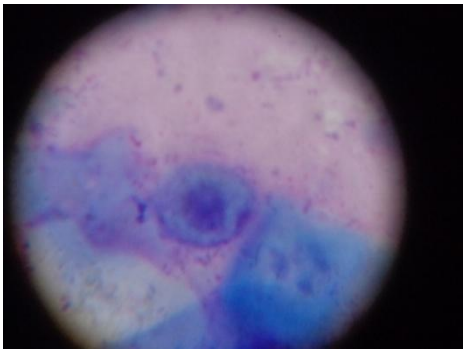


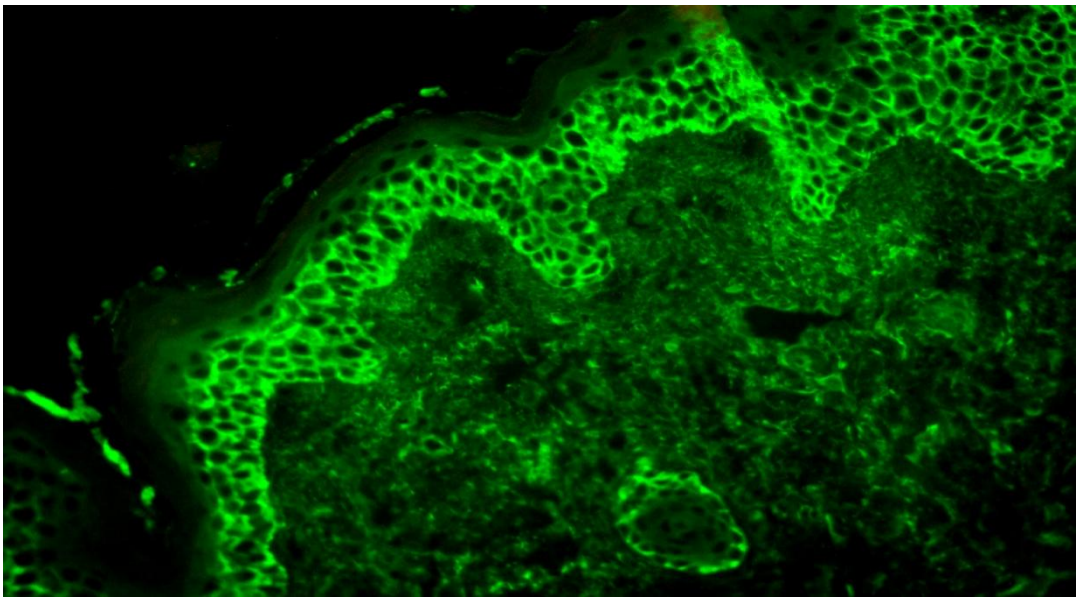
FIG 29: Showing spongiosis in a case of eczema



(A)



(B)



(C)

FIG 30: Pemphigus foliaceus
(A) A subcornial bullae with acantholytic cells and neutrophils in cavity
(B) Acantholytic cell in tzank smear
(C) The intercellular IgG deposits in intercellular space of epidermis

DISCUSSION

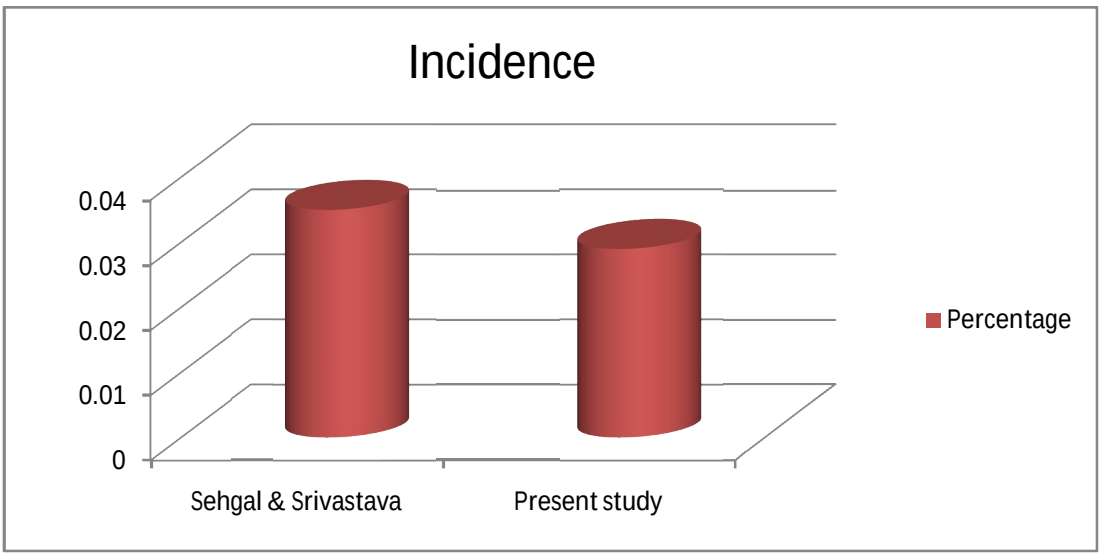
The diagnosis of erythroderma was made based on morphology and percentage of involvement of skin.

INCIDENCE

In our study, the average annual incidence of erythroderma in patients with skin diseases was 0.029 % attending our OPD. The incidence of erythroderma noted in the study done by Sehgal & Srivastava[9] in 1986 was 0.035%.

Table No.10: Incidence of erythroderma in various studies

Study	Incidence
Sehgal & Srivastava[9] -1986	0.035 %
Present study	0.029 %



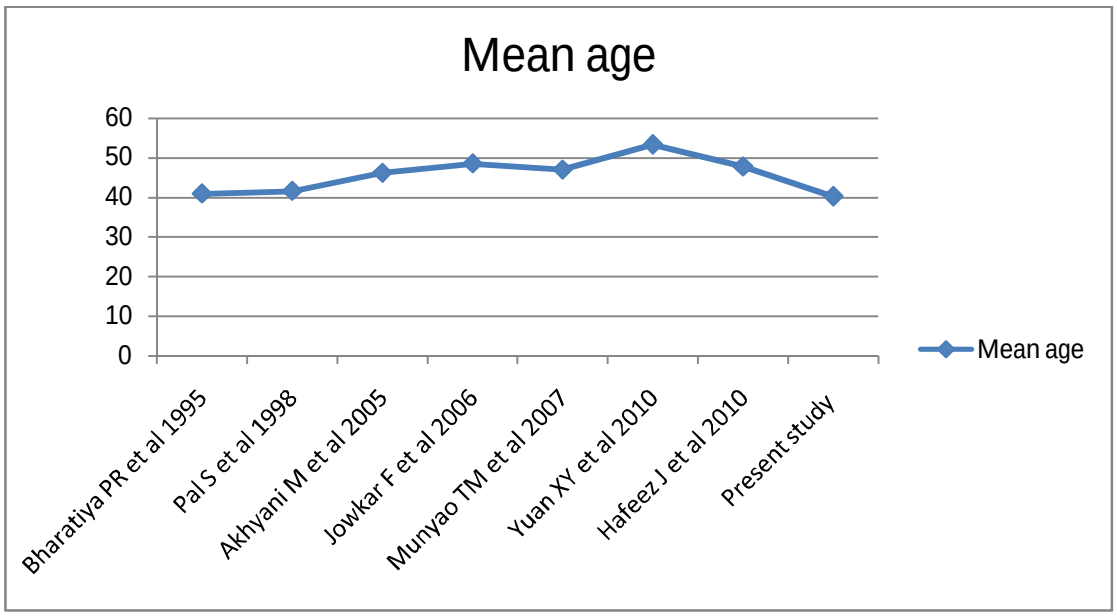
AGE & SEX INCIDENCE

In the study, the maximum number of patients with erythroderma were seen in the age group 50-59 (21.53%) followed by 60-69 (16.92%) and 40-49 (13.84%).

This is in concurrence with other studies, where the maximum incidence was seen in 50-59 years of age group[3,27,96]. The mean age of the patient in the study being 40.25 years.

Table No.11: Mean age of erythroderma in various studies

Author	**Mean age**
Bharatiya PR[27] et al 1995	40.93
Pal S[3] et al 1998	41.6
Akhyani M[26] et al 2005	46.2
Jowkar F[97] et al 2006	48.6
Munyao TM[98] et al 2007	47
Yuan XY[25] et al 2010	53.4
Hafeez J[96] et al 2010	47.8
Present study	40.25

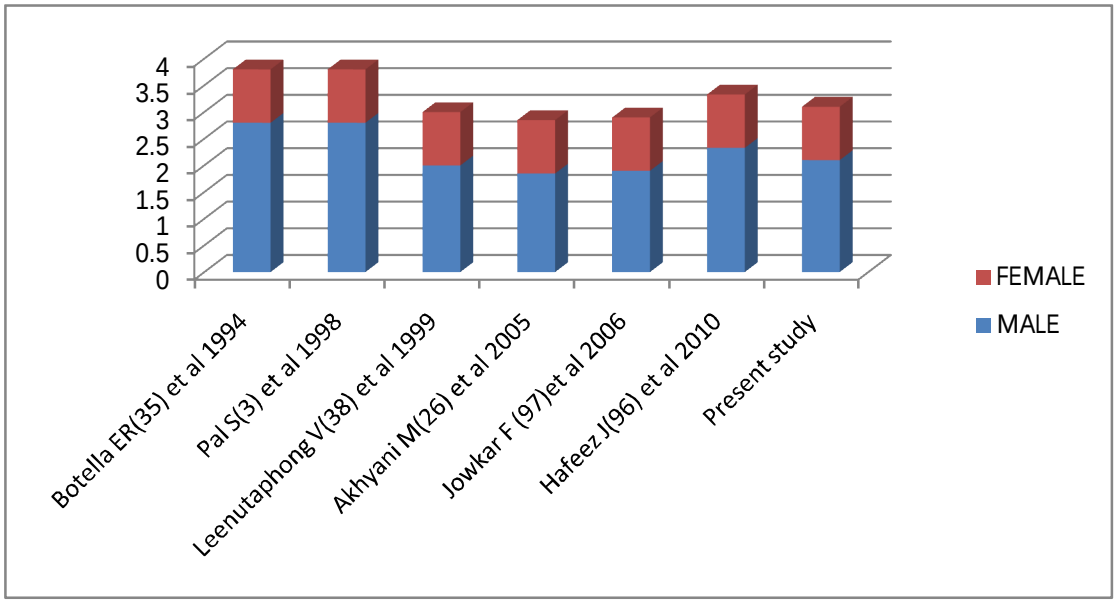


Previous studies have shown a male predominance with male to female ratio approximately between 1.85:1 to 2.8:1. Our study also have shown a male predominance with male to female ratio being 2.1:1.

Table No.12: Sex ratio of erythroderma

Studies	Male to female ratio
Botella ER[35] et al 1994	2.8:1
Pal S[3] et al 1998	2.8:1
Leenutaphong V[38] et al 1999	2:1
Akhyani M[26] et al 2005	1.85:1
Jowkar F[97] et al 2006	1.9:1
Hafeez J[96] et al 2010	2.33:1
Present study	2.1:1

SEX RATIO



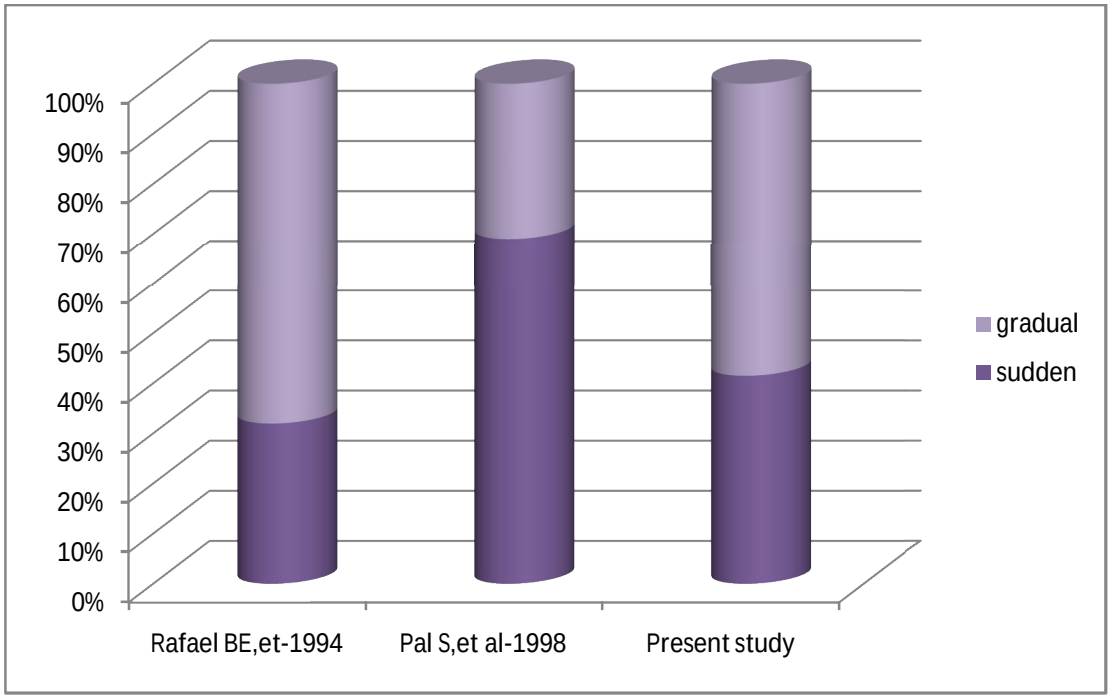
ONSET

Our study showed that 27 patients (41.53%) had acute onset of erythroderma. Among them 17 patients were drug induced erythroderma, 5 were psoriasis, 2 were phytophotodermatitis and one each of pemphigus foliaceous, seborrheic dermatitis and idiopathic.

Remaining 38 (58.46%) patients had a rather insidious onset with a gradually progressing course. An acute onset was seen in 32% of patients in a study done by Rafael BE et al[28] and 69% on a study conducted by Pal S et al[3] .

Table No.13: percentage of sudden onset of erythroderma in different studies

Authors	Percentage
Rafael BE, et al - 1994	32
Pal S, et al - 1998	69
Present study	41.53



CLINICAL FEATURES

Irrespective of etiology, clinical features of erythroderma were almost identical. Once the erythroderma is fully established it is difficult to differentiate between various causes of erythroderma. Scaling and

erythema was seen in all patients, in most cases it was erythema which begins first followed by scaling over a period of 4-5 days. In acute cases the scales were large and in chronic cases they were of small size.

In our study, commonest symptoms were generalised scaling (100%), redness (100%) followed by itching (64.62%), malaise (61.54%), chills (47.69%), fever (27.69%) and oliguria (3.07%). None of the patients had dermatogenic enteropathy. In studies done by Hafeez et al[96], Akhyani M[26] et al, Yuan XY [25]et al, Bandyopathyay D[31] et al, also shown generalized scaling and erythema in upto 100% of cases.

Hyperthermia was noted in 27.69% of patients. None of the patients had hypothermia. The study by Pal and colleagues[3] had shown hyperthermia in 40% and hypothermia in 5.5% of patients.

Lymphadenopathy was seen in 32.30% of cases. Most commonly inguinal lymph nodes were involved followed by axillary lymph nodes. Lymph node biopsy was done in 3 patients, out of which 1 patient showed features suggestive of NHL and other 2 patients showed features of dermatopathic lymphadenopathy. In a study by Pal and colleagues[3] lymphadenopathy was present in 55.50% of cases and all were dermatopathic lymphadenopathy except in one case it was Hodgkin's

lymphoma. Other studies have shown lymphadenopathy ranging from 10% to 67.40%.[25,26,27,31,39,96,97]

Pedal edema of pitting type was observed in 40% of cases. Pedal edema has been reported in 14.4% to 78.67% of cases in various studies.[25,26,27,31,39,96,97]

Mucosal involvement was observed in 9.23% of cases in the form of oral mucosal congestion and erosions. Other studies have shown mucosal involvement ranging from 1% to 36.6% of cases.[3,25,26]

Hepatomegaly was seen in 2 patients (3.07%). One patient had NHL and the other had dapsone syndrome. Similar finding was seen in a study done by Yuan XY and colleagues.[25]

The “nose sign” of erythroderma was reported by Pavithran.[61] In our study it was present in 31 cases(47.69%). The reason for this phenomenon is not exactly known but it is suggested that it is either due to greater exposure of nose to sunlight , with its presumptive anti mitotic activity or the habit of frequent rubbing of nose leading to removal of scales.

In our study, deck chair sign was seen in 7 patients (10.76%). Pal S and colleagues[3] had described this sign in 5.5% of their patients.

Hair loss was observed in 23.07% cases in our study. It was observed in 24% of cases in a study by Sudho and colleagues[39] and in 30% of cases by Pal and colleagues[3]

Nail changes was observed in 69.23% of cases. Most common nail change was ridging of nails (26.15%) followed by subungual hyperkeratosis (18.46%). The study done by Sudho and colleagues[39] had shown pitting (24%) and onycholysis (24%) to be the most common nail changes.

Table No.14: Nail changes in erythroderma

Nail changes	**Sudho et al[39]**	**Present study**
Pitting	24%	16.92%
Onycholysis	24%	9.23%
Beau's line	20%	12.30%
Subungual hyperkeratosis	16%	18.46%
Ridges	16%	26.15%
Nail discolouration	12%	9.23%
Shiny nails	8%	9.23%

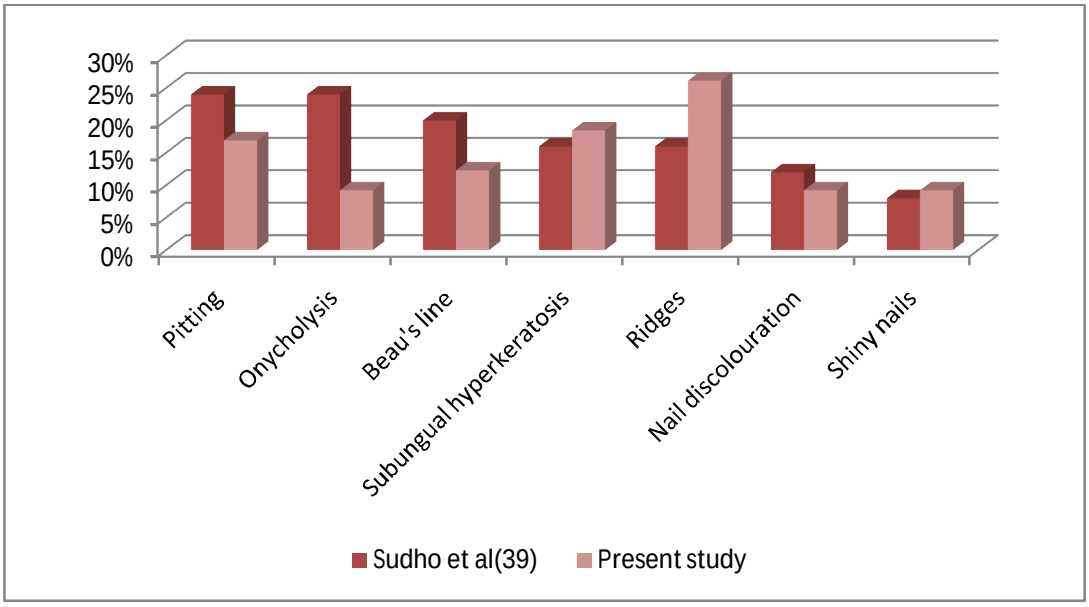
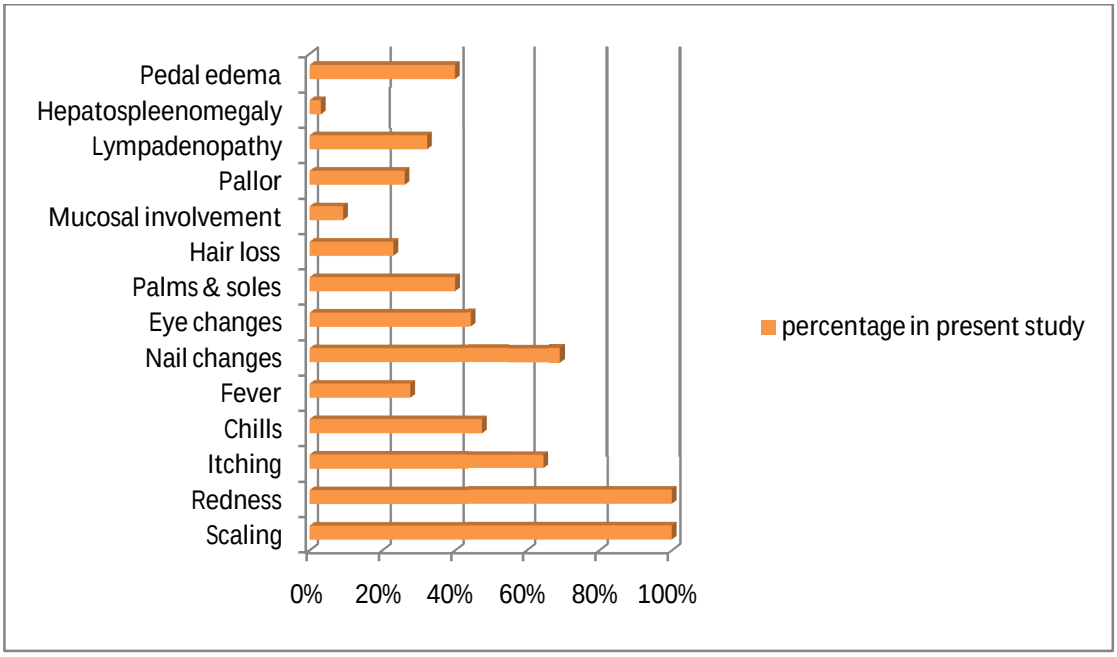


Table No.15: comparison of clinical features of erythroderma in various studies

Clinical features	Hafeez J[96] et al 2010	Jowkar F[97] et al 2006	Akhyani M[26] et al 2005	Bharatiya PR[27] et al 1995	Yuan XY[25] et al 2010	Bhadyopadhyay D[31] et al 1999	Sudho R[39] et al 2003	Pal S[3] et al 1998	Present study
Scaling	100%	56.8%	100%	100%	100%	100%	100%	84.4%	100%
Redness	100%	100%	100%	86.95%	100%	100%	80%	100%	100%
Itching	96%	-	97.5%	-	93.9%	-	80%	-	64.62%
Chills	-	30.3%	-	-	18.3%	-	60%	-	47.69%
Fever	30%	4.9%	33.6%	-	37.8%	-	32%	40%	27.69%
Nail changes	40%	36.2%	-	54.35%	336.6%	56%	64%	-	69.23%
Eye changes	-	-	-	-	-	-	16%	-	44.61%
Palms & soles	-	8.8%	-	39.13%	-	29.33%	-	-	40%
Hair loss	8%	9.8%	-	-	-	-	24%	30%	23.07%
Mucosal involvement	2%	-	1%	-	-	2.66%	32%	36.6%	9.23%
Pallor	10%	-	-	80.43%	-	-	-	-	26.15%
Lympadenopathy	10%	18.6%	21.3%	67.40%	15.9%	22.67%	32%	55.5%	32.30%
Hepatosplenomegaly	4%	0.9%	4%	15.21%	3.6%	13.33%	8%	25.5%	3.07%
Pedal edema	14%	27.4%	14.4%	54.35%	25.60%	78.67%	36%	_	40%

CLINICAL FEATURES



LABORATORY FINDINGS

Hemoglobin was low in 26.15% of cases. In a study by Bandyopadhyay[31] and colleagues it was observed in 48% of cases and in 4.90% of cases in Jowkar F[97] and colleagues series.

Eosinophilia was seen in 12.30% of cases which is similar to the study by Bharatiya PR and colleagues.[27]

Increased ESR was seen in 30.76% of cases in our study. In a study by Jowkar F and colleagues[97] it was observed in 20.58% of cases.

Hypoproteinemia was observed in 12.30% of cases in our study which is in concurrence with the study done by Yuan XY et al[25]

(13.4%). Increased serum creatinine was observed in 1.53% of cases. None of the patient had significant electrolyte imbalance.

ETIOLOGY OF ERYTHRODERMA

Psoriasis was the most common cause accounting for 30.76% of cases. Similar findings were noted in studies done by Sudho et al[39] (32%) and Bandyopadhyay et al[31](33.33%). Diagnosis of psoriasis was made on the basis of prior history of being treated for psoriasis, clinical examination and histopathological correlation.

Eczema as a cause of erythroderma was seen in 16.92% of cases which is in concurrence with the study done by Bandhyopadyay and colleagues (17.33%).[31]

Ichthyosis as a cause was observed in 6.15% of cases. In a study done by Pal S and colleagues[3] 7.8% of cases had ichthyosis as a cause of erythroderma.

PRP was seen in 3.07% of cases, which was correlating with the study by Pal S and colleagues.[3]

Pemphigus foliaceous as a cause was observed in 3.07%, which is in concurrence with the study done by Sudho et al[39] (4%) and Hafeez et al[96] (4%).

Crusted scabies was seen in 1.53% of cases, which was correlating with the previous studies. [3,31,96]

Drug reaction as etiology was seen in 26.15% of cases. Drugs implicated were carbamazepine, native medicine, ART, ATT, sodium valoporate, dapsone, ofloxacin and uncommon drugs which coincides with the previous studies.[39,101] Most common drug that lead to erythroderma was carbamazepine (5 out of 17 cases - 29.41%) as described in study by Mapar MA and colleagues[101] (8 out of 20 cases) and Akhyani M et al [26] (12 out of 21 cases). A recent history of intake of newer drug, sudden onset, peripheral eosinophilia and improvement of the condition once the offending drug was withheld helped us for implicating drug reaction as the etiology of erythroderma.

Malignancy was seen in 1 patient (1.53%), the underlying malignancy being NHL which was diagnosed by peripheral smear examination and lymphnode biopsy. In various previous studies, malignancy as a cause of erythroderma ranged from 2.9% to 29% of cases.

In 10.76% of cases, eiological factor for erythroderma could not be determined inspite of thorough clinical and histopathological examinations. The incidence of idiopathic erythroderma ranged from

6.1% to 32% in various previous studies.[25,30,101] Repeated biopsies in two patients did not yield any help in the diagnosis. The other patients did not give consent for the same. It is imperative to have long term follow up and to rule out cutaneous lymphoma as the cause.

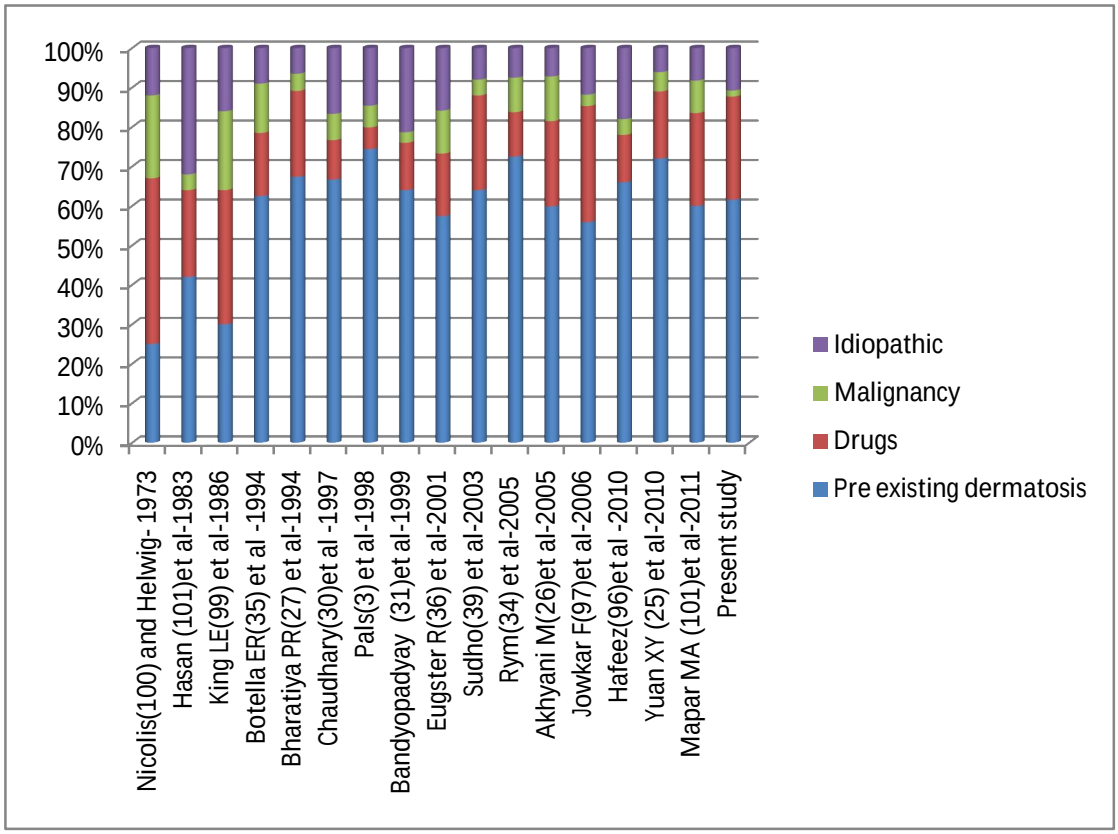
Among the 53 patients on whom the biopsy was done, the histopathology helped in correlating and confirming the diagnosis in 43.39% of cases, where as in study by Bandyopadhy and colleagues there was correlation in 52% of cases.

Experience from the study supports the view that erythroderma although a very disturbing disorder, does not pose a significant risk to the patients life. The main challenge lies in identifying the underlying cause and managing accordingly.

Table No.16: Comparison of etiology of erythroderma in various studies

Study	Pre exsisting dematosis								Drugs	Malignancy	Idiopathic
	Psoriasis	Eczema	Icthyosis	P.F	PRP	N.S	Others	Total			
Nicolis[100] and Helwig- 1973	-	-	-	-	-	-	-	25	42	21	12
Hasan [101]et al-1983	10	30	2	0	0	0	0	42	22	4	32
King LE[99] et al-1986	-	-	-	-	-	-	-	30	34	20	16
Botella ER[35] et al -1994	-	-	-	-	-	-	-	62.5	16	12.5	9
Bharatiya PR[27] et al-1995	-	-	-	-	-	-	-	67.40	21.74	4.35	6.51
Chaudhary[30]et al -1997	40	26.66	0	0	0	0	0	66.66	10	6.66	16.66
Pals[3] et al-1998	37.8	12.2	7.8	5.6	2.2	2.2	6.6	74.4	5.5	5.5	14.6
Bandyopadyay [31]et al-1999	33.33	17.33	1.33	5.33	5.33	1.33	0	63.98	12	2.67	21.33
Eugster R[36] et al-2001	-	-	-	-	-	-	-	58	16	11	16
Sudho[39] et al-2003	32	20	0	4	0	0	8	64	24	4	8
Rym[34] et al-2005	51.25	7.5	0	6.25	1.25	1.25	5	72.50	11.25	8.75	7.5
Akhyani M[26]et al-2005	27.8	19.6	1	1	8.2	1	1.2	59.8	21.6	11.3	7.2
Jowkar F[97]et al-2006	19.60	34.31	0	0	0.98	0.98	0	55.9	29.4	2.9	11.8
Hafeez[96]et al -2010	16	38	4	4	2	2	0	66	12	4	18
Yuan XY [25] et al-2010	-	-	-	-	-	-	-	72	17	4.9	6.1
Mapar MA [101]et al-2011	21.17	32.94	2.35	1.17	0	0	2.34	59.97	23.52	8.23	8.23
Present study	30.76	16.92	6.15	3.07	3.07	1.53	0	61.53	26.15	1.53	10.76

Comparison of etiology of erythroderma in various studies

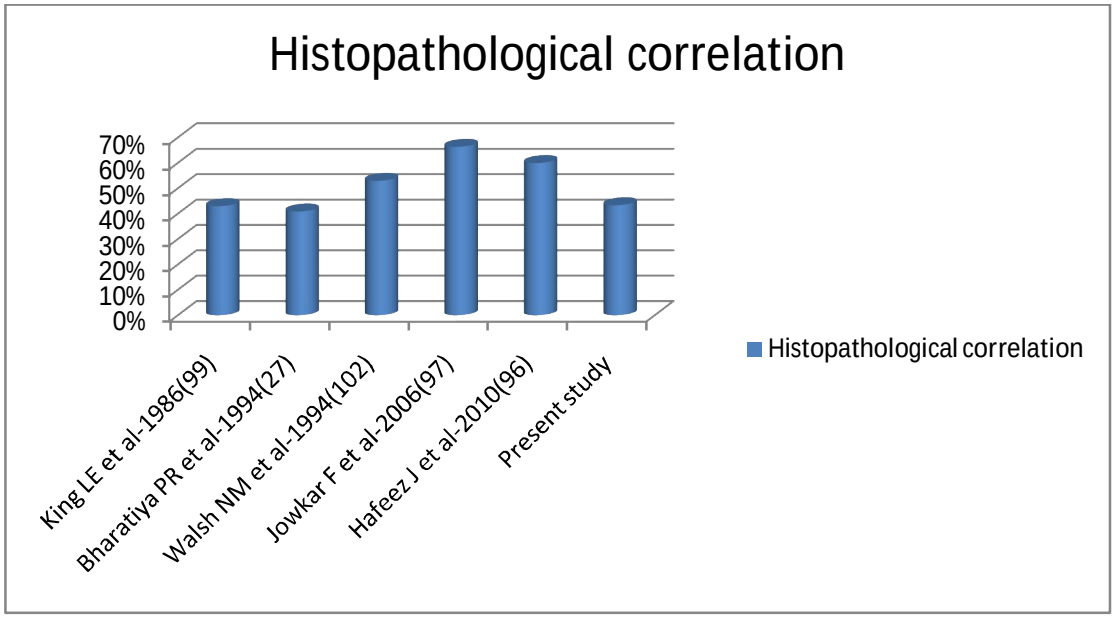


HISTOPATHOLOGICAL CORRELATION

In this study skin biopsy was done in 53 patients and the histological findings were compared with the clinical diagnosis. Among this 43.39% of biopsy section was rewarding in arriving at a diagnosis. In various studies histopathological correlation ranged between 40.91% to 66.4%. [27,96,97]

Table No.17: Histopathological correlation in various studies

Study	Histopathological correlation
King LE et al-1986[99]	43%
Bharatiya PR et al-1994[27]	40.91%
Walsh NM et al-1994[102]	53%
Jowkar F et al-2006[97]	66.40%
Hafeez J et al-2010[96]	60%
Present study	43.39%



SUMMARY AND CONCLUSION

After analysing 65 cases of erythroderma admitted in our hospital during a period of two years the following observations were made;

- The average annual incidence of erythroderma among the patients who attended the skin department of our hospital was 0.029%.
- Maximum number of patients were in the age group 50-59 (21.53%) with a male to female ratio of 2.1:1
- The onset was acute in 41.53% and gradual in 58.46% of cases.
- Itching (64.62%) was the most common symptom followed by malaise (61.54%), chills (47.69%), pedal edema (40%), palms & soles involvement (40%) and fever (27.69%).
- Lymphadenopathy was observed in 32.30% of cases.
- Hyperthermia was seen in 27.69% of cases, pallor in 26.15%, diffuse hair loss in 23.07% and hepatosplenomegaly in 3.07% of cases.

- Nail changes was observed in 69.23% of cases, ridging (26.15%) being the most common nail change noted.
- Eye involvement was observed in 44.61% of cases.
- Mucosal involvement was observed in 9.23% of cases and all of them were drug induced erythroderma.
- Eosinophilia and hypoproteinemia was seen in 12.30% of cases, altered liver function test in 7.69% of cases and raised level of serum creatinine in 1.53% of cases.
- None of the patients had electrolyte imbalance and metabolic complications.
- Atypical lymphocytes in peripheral blood smear was seen in a case of Non Hodgkin's lymphoma.
- The most common etiological factor was pre existing dermatosis (61.53%) followed by drug reactions (26.15%), idiopathic (10.76%) and malignancy (1.53%).
- Among the pre existing dermatosis, psoriasis (30.76%) was the leading cause followed by eczema (16.92%), ichthyosis (6.15%), pemphigus foliaceous (3.07%), pityriasis rubra pilaris (3.07%) and crusted scabies (1.53%).

- Clinical features were almost identical in nearly all patients.
- Histopathology was rewarding in 43.49% of cases in whom skin biopsy was performed.
- Drug induced erythroderma had a good prognosis and erythroderma due to Non Hodgkin's lymphoma had a worst prognosis.

Although no etiological diagnosis could be established in a number of cases it is imperative to follow up and investigate the case periodically since idiopathic erythroderma may turn to cutaneous lymphoma in the long run in a percentage of cases.

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PROFORMA

Name Address

Age Occupation

Sex

CHIEF COMPLAINTS:

Itching, burning, shivering

Redness of skin, scaling

Hair lose, nail changes, oral ulcers

HOPI:

Mode of onset (acute or chronic)

Duration of illness

Course of illness

Remissions and exacerbations

Pruritus

Tightness of skin

Photosensitivity

Contact with plants

Joint pain

Fever, malaise, chills

Decreased urine output / leg swelling/ diarrhoea

Breathlessness/ palpitation

Topical applications/ indigenous medicines, drug intake

Seasonal variation, stress, emotional disturbancess

Focal sepsis , atopy

Suggestive of pre existing skin lesions

PAST HISTORY

Duration of pre existing skin disease (onset, duration, distribution and course of disease.

Similar episodes in the past

Diabetes , hypertension , liver disease , ischemic heart disease.

TREATMENT HISTORY

Drug intake prior to the onset of present illness

Treatment for the present illness elsewhere

Drug taken/withdrawn for preexisting skin disease

PERSONAL HISTORY

Alcohol , smoking

FAMILY HISTORY

Consanguinity in parents

Family members with ichthyosis , immunodeficiency

CLINICAL EXAMINATION

GENERAL EXAMINATION

pallor

Icterus

Clubbing, cyanosis

Pedal edema

Lymphadenopathy (site, size, tenderness, mobility,discrete /matted)

VITAL SIGNS

- Temperature
- Pulse
- Blood pressure
- Signs of dehydration

SYSTEMIC EXAMINATION

- Cardiovascular system
- Respiratory system
- Abdomen
- Central nervous system

Per rectal examination

DERMATOLOGICAL EXAMINATION

- Skin erythema
- Scaling , types of scaling
- Areas of involvement and sparing if any
- Skin thickening, lichenification and flaking
- Clinical clues of preexisting skin disease.
- Scalp- hair loss/ scales
- Nail changes
- Eye changes
- Ectropion of eye lids
- Mucosal involvement

CASE NO	AGE	SEX	ONSET	DURATION	H/O UD/D	R/S	I	C/R	TEMP	PE	L	P/S	SCALP	NAILS	EYES	CBC	BIOCHEM	SKIN/LN
1	57	M	G	3 MON	PSORIASIS	+	+	+	39	+	-	II	SC	SH,D	E	AN,EE	N	CONSIST
2	60	M	G	6 MON	PSORIASIS	+	+	-	N	-	-	N	SC,HL	SH,PI	E	AN,EE	N	CONSIST
3	30	M	G	1YR	IDIOPATHIC	+	+	-	N	-	+	SC,PPK	SC	PI,RI	N	N	HP	DERMAT
4	21	M	S	10DAYS	T. CBZ	+	-	+	38.8	+	-	N	N	N	N	N	N	DERMAT
5	60	M	G	2 MON	IDIOPATHIC	+	+	-	N	-	+	N	N	BL	E	AN	N	DERMAT
6	58	M	S	1WK	DRUG	+	-	+	38.5	-	-	PL	N	N	C	EO	N	DERMAT
7	2	F	G	1YR	AD	+	+	-	N	-	-	N	N	N	N	EO	N	-
8	63	M	G	6MON	PSORIASIS	+	+	-	N	+	+	II	SC	PI,OL,BL	N	EE	N	DERMAT
9	49	F	G	10MON	PSORIASIS	+	+	-	N	-	-	PPK	SC,F	D	C	EE	HP	CONSIST
10	56	M	G	3YR	IDIOPATHIC	+	+	-	N	+	-	N	HL	RI	N	AN,EE	HP	DERMAT
11	31	F	S	12DAYS	PF	+	-	+	N	+	-	N	CR	OM,RI	C	AN,EE	N	CONSIST
12	12	F	G	2YR	PSORIASIS	+	+	+	N	-	+	FIS,SC	N	PI,SH	N	N	N	CONSIST
13	63	M	G	20DAYS	ABCD	+	+	+	38	+	+	N	HL	SN	N	AN,EO	N	DERMAT
14	56	M	S	3MON	PSORIASIS	+	+	-	N	+	+	II	SC	SH,D,OL	N	N	N	DERMAT
15	58	M	G	6MON	PP	+	+	+	39	-	-	N	SC, HL	D, BL	E	AN	ELE	DERMAT
16	2	FCH	G	SINCE BIRTH	NBIE	+	-	-	N	-	-	-	N	N	N	N	N	-
17	50DAYS	FCH	G	1MON	SD	+	-	-	N	-	-	N	SC	N	N	N	N	-
18	71	M	G	2MON	PPD	+	+	+	38.5	+	+	N	HL	SN	N	AN,EO,EE	N	DERMAT

19	58	M	G	3MON	IDIOPATHIC	+	+	+	N	+	+	N	N	SN,D	E	EE	N	DERMAT
20	55	M	S	3WK	PPD	+	+	-	N	-	-	N	N	SN,D,SH	N	N	HP	DERMAT
21	60	M	S	10DAYS	NATIVE MED	+	+	+	39	-	-	N	N	N	C	N	N	DERMAT
22	42	M	S	12DAYS	T.CBZ	+	-	+	38.8	-	-	PL	N	N	N	N	N	CONSIST
23	60	M	G	26DAYS	PSORIASIS	+	+	+	N	+	+	N	SC	PI,RI	N	N	N	CONSIST
24	50	F	S	10DAYS	T.CBZ	+	-	+	N	-	+	N	N	BL	C	N	ELE	DERMAT
25	40	F	G	1MON	NS	+	+	+	N	+	+	CR	HL	SH,D,BL	N	AN,EE	N	-
26	29	M	S	12DAYS	DRUG	+	-	+	N	-	-	N	N	N	N	N	N	DERMAT
27	60	M	G	6MON	PPD	+	+	-	N	+	+	N	HL	RI	DMF	AN	N	DERMAT
28	20	M	S	2WK	VALPROATE	+	-	+	39.4	-	-	N	N	N	N	N	ELE	CONSIST
29	41	M	G	6MON	PRP	+	+	-	N	-	-	PPK	HL	N	E	N	N	CONSIST
30	25	M	S	2WK	T.CBZ	+	+	+	N	-	-	PL	N	N	N	N	N	DERMAT
31	61	M	G	1YR	PSORIASIS	+	+	-	N	-	-	N	SC,HL	SH,RI	E	AN	HP	CONSIST
32	17	F	S	2WK	PP	+	-	+	39	-	-	N	SC	PI,RI,OM	N	N	N	CONSIST
33	2mon	MCH	S	1MON	SD	+	-	-	N	-	-	N	SC	N	N	N	N	-
34	10	MCH	G	SINCE BIRTH	LI	+	-	-	N	-	-	SC	SC	SH	E	N	N	-
35	28	F	S	1WK	ART	+	+	+	38.6	-	+	PL	HL	BL,RI	N	AN,EE,EO	ELE	DERMAT
36	18	F	G	7MON	IDIOPATHIC	+	+	-	N	+	-	PPK	HL,SC	OL,SH	E	N	N	DERMAT
37	8	MCH	S	1MON	PSORIASIS	+	+	-	N	-	-	II	SC	PI,BL	N	N	N	CONSIST
38	35	F	S	3WK	DAPSONE	+	-	+	39	+	+	N	N	N	I	N	ELE	DERMAT
39	48	M	G	1MON	PSORIASIS	+	+	-	N	+	-	N	SC	D,SH	N	N	N	CONSIST

40	37	M	G	3MON	PRP	+	+	-	N	-	-	PPK	N	RI	N	N	N	CONSIST
41	5mon	FCH	G	SINCE BIRTH	LI	+	-	-	N	-	-	N	N	N	E	N	N	-
42	53	M	G	6MON	NHL	+	+	-	N	+	+	FIS,PPK	HL	D,SH,OM	N	TC74000,LB-68%	N	LN BX-NI
43	23	F	S	1WK	OFLOX	+	+	+	39.5	-	-	PL	N	N	N	N	N	DERMAT
44	29	M	S	2WK	ART	+	+	+	N	-	+	N	N	N	N	AN	N	-
45	4	FCH	G	3MON	AD	+	-	-	N	-	-	N	N	N	DMF	N	N	-
46	32	F	S	12DAYS	PSORIASIS	+	-	+	N	-	-	SC,FIS	N	N	N	N	N	CONSIST
47	38	M	S	10DAYS	CBZ	+	-	+	39	-	+	PL	N	N	C	EO,EE	N	CONSIST
48	43	M	G	2MON	PSORIASIS	+	+	-	N	+	-	FIS,SC	SC	SH,OM	N	N	N	CONSIST
49	41	M	S	5DAYS	NATIVE MED	+	+	+	N	-	-	N	N	N	C	AN	N	DERMAT
50	52	M	G	3MON	PSORIASIS	+	+	-	N	+	-	N	N	RI	N	AN,EE	N	CONSIST
51	59	M	S	2WK	PPD	+	+	+	N	+	+	N	N	SN,RI	C	EE	N	DERMAT
52	28	M	S	10DAYS	NATIVE MED	+	+	+	38.5	+	-	PL	N	N	N	EO,EE	N	DERMAT
53	66	F	G	2MON	PSORIASIS	+	+	-	N	+	-	N	SC	SN	E	EE	HP	DERMAT
54	46	F	S	1WK	DRUG	+	-	+	39	+	+	N	N	N	C	EE	N	DERMAT
55	60	F	G	1MON	ABCD	+	+	-	N	-	-	N	HL	SN,R	N	EE	N	DERMAT
56	44	M	G	1MON	PSORIASIS	+	-	-	N	-	-	FIS	SC	OL,SH,RI	N	AN,EE	N	CONSIST
57	1	FCH	G	SINCE BIRTH	LI	+	-	-	N	-	-	N	SC	N	E	N	N	-
58	60	M	G	4MON	IDIOPATHIC	+	+	-	N	+	+	N	HL	BL,RI	E,C	EE	HP	DERMAT

59	55	M	S	3MON	IDIOPATHIC	+	+	+	39	+	-	N	N	N	N	EE	N	DERMAT
60	50	M	S	4MON	PSORIASIS	+	-	-	N	-	-	FIS,SC	SC	RI	E	N	N	CONSIST
61	52	M	G	5WK	PF	+	-	+	N	-	-	N	N	RI	C	AN,EE	N	CONSIST
62	31	F	S	2MON	ATT	+	-	-	N	-	+	SC	N	N	N	EO	ELE	-
63	35	F	G	4WK	PSORIASIS	+	+	-	N	+	-	N	SC	PI	N	N	N	CONSIST
64	2MON	M	G	6WK	SD	+	-	-	N	-	-	N	SC	N	N	N	N	-
65	14	M	G	4MON	PP	+	+	+	38.6	+	-	FIS	SC,HL	PI,OL	E	N	HP	CONSIST

KEY TO MASTER CHART

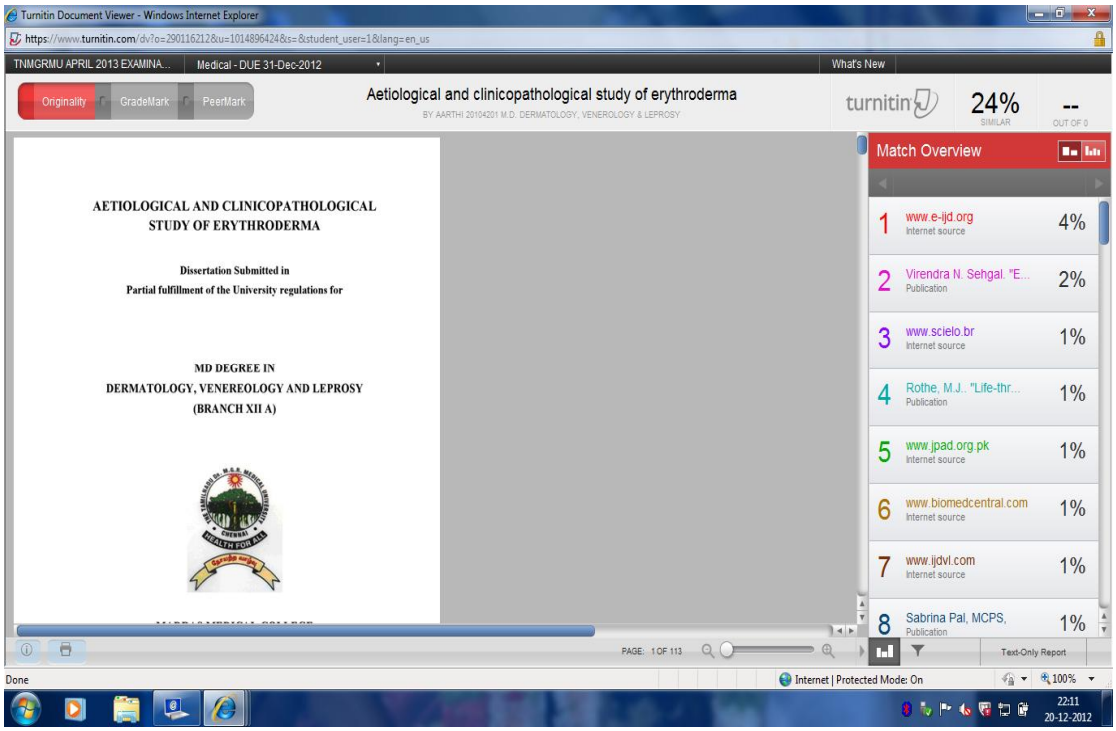
M	:	Male
F	:	Female
MCH	:	Male child
FCH	:	Female child
+	:	Present
_	:	Absent
N	:	Normal
G	:	Gradual
S	:	Sudden
H/O UD,D	:	History of underlying dermatoses, drug intake
AD	:	Atopic dermatitis
PF	:	Pemphigus foliaceous
PP	:	Pustular psoriasis
NBIE	:	Non bullous ichthyosiform erythroderma
SD	:	Seborrheic dermatitis
PPD	:	Phytophotodermatitis
ABCD	:	Air born contact dermatitis
NS	:	Norwegian scabies
NHL	:	Non Hodgkin’s lymphoma
PRP	:	Pityriasis rubra pilaris
OFLOX	:	Ofloxacin
CBZ	:	Carbamazepine

ART	:	Antiretroviral therapy
ATT	:	Antituberculous treatment
R/S	:	Redness and scaling
I	:	Itching
C/R	:	Chills/Rigor
TEMP	:	Temperature
PE	:	Pedal edema
L	:	Lymphadenopathy
P/S	:	Palms and soles involvement
II	:	Instep involvement
SC	:	Scaling
PPK	:	Palmoplantar keratoderma
PL	:	Peeling
FIS	:	Fissuring
CR	:	Crusting
HL	:	Hair loss
SH	:	Subungual hyperkeratosis
D	:	Onychodystrophy
PI	:	Pitting
RI	:	Ridging
OL	:	Onycholysis
BL	:	Beau’s line
OM	:	Onychomadesis
SN	:	Shiny nails

C	:	Congestion
E	:	Ectropion
I	:	Icterus
DMF	:	Denny morgon fold
CBC	:	Complete blood count
AN	:	Anemia
EO	:	Eosinophilia
LB	:	Lymphoblast
TC	:	Total count
EE	:	Elevated erythrocyte sedimentation rate
BIOCHEM	:	Biochemical investigations
HP	:	Hypoproteinemia
ELE	:	Elevated liver enzymes
TZ	:	Tzank smear
REC	:	Recurrence
SCR	:	Scrapping for fungus, acarus
PR	:	Parthenium
K	:	Potassium dichromate
LN BX	:	Lymph node biopsy
DIF	:	Direct immunofluorecence

ABBREVIATIONS

CTCL	Cutaneous T cell lymphoma
PRP	Pityriasis rubra pilaris
HIV	Human immunodeficiency virus
PF	Pemphigus foliaceous
NS	Norwegian scabies
IgE	Immunoglobulin E
OPD	Out patient department
ART	Antiretroviral therapy
ATT	Antituberculous treatment
NHL	Non Hodgkin's lymphoma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CERTIFICATE OF APPROVAL

To
Dr. Aarthi M
PG in MDDVL
Madras Medical College, Chennai -3

Dear Dr. Aarthi M

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Aetiological and clinicopathological study of Erythroderma" No. 08122011

The following members of Ethics Committee were present in the meeting held on 22.12.2011 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan. MD -- Chairperson
2. Prof. R. Nandhini MD -- Member
   Director, Institute of Pharmacology ,MMC, Ch-3
3. Prof. Pregna B. Dolia MD -- Member
   Director , Institute of Biochemistry, MMC, Ch-3
4. Prof. S. Regunanthan, MD -- Member
   Prof of Internal Medicine, MMC, Ch-3
5. Prof. Md Ali MD. DM -- Member
   Prof & Head , Dept. of MGE, MMC,Ch-3
6. Thiru. S. Govindsamy. BA BL -- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee